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A CROSS-SECTIONAL STUDY OF SELECTED MODIFIABLE CARDIOVASCULAR RISK FACTORS AMONG SALES EXECUTIVES IN AN URBAN ORGANIZED SECTOR INDUSTRIAL POPULATION IN CHENNAI

Dissertation submitted for
The Tamil Nadu Dr. M.G.R. Medical University,
Chennai.

M.D. DEGREE EXAMINATION
BRANCH- XV
(COMMUNITY MEDICINE)

MADRAS MEDICAL COLLEGE
CHENNAI-600 003.

MARCH 2009

Certificate

Certified that this dissertation is the bonafide work of ***Dr.*** **KUMAR CAROLINE PRIYA** *on* "A CROSS–SECTIONAL STUDY OF SELECTED MODIFIABLE CARDIOVASCULAR RISK FACTORS AMONG SALES EXECUTIVES IN AN URBAN ORGANIZED SECTOR INDUSTRIAL POPULATION IN CHENNAI" *during her M.D. (Community Medicine) course from May 2006 to March 2009 at the Madras Medical College and Government General Hospital, Chennai.*

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DECLARATION

*I solemnly declare that this dissertation entitled "*A CROSS–SECTIONAL STUDY OF SELECTED MODIFIABLE CARDIOVASCULAR RISK FACTORS AMONG SALES EXECUTIVES IN AN URBAN ORGANIZED SECTOR INDUSTRIAL POPULATION IN CHENNAI*" was done by me at Institute of Community Medicine, Madras Medical College, during 2006-2009 under the guidance and supervision of* ***Prof. S. ELANGO****,* ***M.D****. This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree in Community Medicine (Branch-XV).*

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ACKNOWLEDGEMENTS

"Ask and you will receive seek and you will find".

For the completion of any work, guidance and support is essential. I would like to take this opportunity to thank each person for the help I received in completing this study.

I would like to thank ***Dr. T.P. Kalanithi,*** *Dean, Madras Medical College and Research Institute, Government General Hospital, Chennai, for permitting me to do this study.*

I gratefully acknowledge and sincerely thank Dr. S. Elango, Director, Institute of Community Medicine, Madras Medical College, Chennai, for his valuable guidance and support.

I wish to thank my Associate and Assistant Professors, Dr. R. Nagarani, Dr.Arunmozhi and Dr. V.V. Anantharaman for their encouragement and advice.

I am grateful to Mrs. Indra Subramaniyam, Director, Ehrlich Laboratory, Chennai-14, and Dr. Pragna B. Dolia Director, Institute of Biochemistry, Madras Medical College, Chennai, for helping me in the Laboratory estimation.

I am deeply thankful to Dr. Prabhdeep Kaur, ICMR, Anannur, and Dr. Stanley Michael, for providing me expert guidance and inspiring me to carry out this study.

I wish to thank Mr.Venkatesh, biostatistician Madras Medical College.

I am thankful to my faculty and my colleagues for their support.

A special thanks to my husband, children and family for being there for me when I needed them the most.

Last but not least I would like to thank the participants in this study for their valuable time and co-operation so that I could bring this study to a fruitful conclusion.

INDEX

ABBREVIATIONS

BMI	Body Mass Index
CHD	Coronary Heart Disease
CVD	Cardiovascular Diseases
DALY	Disability Adjusted Life Years
DBP	Diastolic Blood Pressure
HDL	High-Density Lipoprotein
ICMR	Indian Council of Medical Research
IDDM	Insulin-Dependent Diabetes Mellitus
ISH	International Society of Hypertension
JNC 7	Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and the Treatment of High Blood Pressure
LDL	Low-Density Lipoprotein
NCDs	Non-Communicable Diseases
NIDDM	Non-insulin dependent diabetes mellitus
SBP	Systolic Blood Pressure
WHO	World Health Organization

INTRODUCTION

"MOVE FOR HEALTH" was the World Health Organization theme for the year 2002. In most parts of the world, non-communicable diseases have become a major epidemic. This is due, in part, to a rapid change in lifestyles leading to reduced physical activity, changing diets and increased tobacco use. This trend is present in all societies, rich and poor, developed and developing.[1]

The origin of the current epidemic of Cardiovascular Disease (CVD) can be traced back to the time of industrialization in the seventeen hundreds. The three factors responsible for this were an increase in the use of tobacco products, reduced physical activity and the adoption of a diet high in fat, calories and cholesterol. This rising tide of CVD continued into the 20th century.[2]

Statistical projections show that 64 million people will die in 2015; 41 million (64%) of them will die from chronic diseases. This is a 17% increase in chronic disease deaths from 2005 to 2015. In 2005, chronic Non-Communicable Diseases (NCDs) accounted for 60% of all projected deaths worldwide – i.e. an estimated 35 million people died of NCDs. The projected 35 million deaths from chronic diseases is double the number of deaths from all infectious diseases (including Human Immunodeficiency Virus / Acquired Immunodeficiency Syndrome, tuberculosis, and malaria), maternal and perinatal conditions, and nutritional deficiencies combined. Some 80% of the deaths from

NCDs occur in low and middle-income countries. The five major NCDs are heart disease, stroke, cancer, chronic respiratory diseases and diabetes.[3]

Cardiovascular diseases (mainly heart disease and stroke) are responsible for 30% of all deaths. The Major Cardiovascular diseases include.[4]

- Coronary (or ischaemic) heart disease (heart attack)
- Cerebrovascular disease (stroke)
- Hypertension (high blood pressure)
- Heart failure
- Rheumatic heart disease.

The rise of cardiovascular diseases is attributed to a number of factors.

The gradual reduction and elimination of infectious diseases affecting the young and middle-aged has led to longer lives. Longer life expectancy with increased proportions of older individuals in society has brought cardiovascular diseases to the forefront.

Additionally, atherosclerotic diseases are associated with affluent lifestyles. Surplus food plus reduction in habitual physical activity results in obesity, which also encourages hyperlipidemia and hypertension.[5]

The problem has serious impact. The burden of chronic disease:

- has major adverse effects on the quality of life of affected individuals;
- causes premature death;
- and creates large adverse – and underappreciated – economic effects on families, communities and societies in general.

Chronic diseases undermine the macroeconomic development of many countries. Countries will forego billions in national income over the next 10 years as a result of premature deaths caused by heart disease, stroke, and diabetes.

- China: $558 billion
- Russian Federation: $303 billion
- India: $237 billion.[3]

The causes of the main chronic disease epidemics are well established and well known. The most important modifiable risk factors are:

- unhealthy diet and excessive energy intake;
- physical inactivity;
- tobacco use.

According to 'The WHO STEP wise approach'[6] a "risk factor" refers to any attribute, characteristic, or exposure of an individual, which increases the likelihood of developing a non-communicable disease. Surveillance of just eight selected risk factors (Table 1) which reflect a large part of future NCD burden can provide a measure of the success of interventions.

The rationale for inclusion of core risk factors is therefore that:

- they have the greatest impact on NCD mortality and morbidity;
- modification is possible through effective primary prevention;
- measurement of risk factors has been proven to be valid; and
- measurements can be obtained using appropriate ethical standards.

Table 1 - Risk factors common to major non-communicable conditions.[6]

Risk factor	Condition			
	Cardiovascular disease*	**Diabetes**	**Cancer**	**Respiratory conditions****
Smoking	✓	✓	✓	✓
Alcohol	✓		✓	
Nutrition	✓	✓	✓	✓
Physical inactivity	✓	✓	✓	
Obesity	✓	✓	✓	✓
Raised blood pressure	✓	✓		
Blood glucose	✓	✓	✓	
Blood lipids	✓	✓	✓	

* Including heart disease, stroke, hypertension.
** Including chronic-obstructive pulmonary disease and asthma.
Source(6)

The major modifiable risk factors, in conjunction with the non-modifiable risk factors of age and heredity, explain the majority of new events of heart disease, stroke, chronic respiratory diseases and some important cancers. The major causes of chronic diseases are known, and if these risk factors were eliminated, at least 80% of all heart disease, stroke and type-2 diabetes would be prevented; over 40% of cancer would be prevented.[3]

OBJECTIVES

1. To estimate the prevalence of selected modifiable cardiovascular risk factors among sales executives in an industrial population in Chennai.

2. To study the distribution of risk factors across the various age groups.

JUSTIFICATION

1. Cardiovascular Diseases are major contributors to the global burden of chronic diseases with 29.3% of global deaths and 9.9% of total disease burden, in terms of Disability Adjusted Life Years (DALYs) lost, being reported in 2003.[7] Low and middle income countries accounted for 78% and 86%of CVD deaths and DALYs lost respectively, world wide in 1998.[8] This burden of CVD is predicted to increase substantially in developing countries by the year 2020.

2. In India CVD is projected to be the largest cause of death and disability by 2020[9] with 2.6 million Indians predicted to die due to Coronary Heart Disease (CHD) which constitutes 54.1% of all CVD deaths.

3. The burden of CVD is rising in India. In 2003, the prevalence of CHD in India was estimated to be 3-4 per cent in rural areas, and 8-10 per cent in urban areas, among adults older than 20 years, representing a two-fold rise in rural areas and a six-fold rise in urban areas over the past four decades, with a total of 29.8 million affected (14.1 million in urban areas, and 15.7 million in rural areas) according to population-based cross-sectional surveys.[10]

4. Nearly half of these deaths are likely to occur among young and middle age individuals (30-69 years). This is because Indians experience CVD deaths at least a decade earlier than their counterparts in developed countries. This has the potential to adversely affect India's economy with 52% of CVD deaths occurring in those below the age of 70 years compared to 23% in countries in established market economies.[11]

5. The huge burden of CVD in Indian subcontinent is the consequence of the large population and high prevalence of cardiovascular risk factors.[12]

6. These risk factors include smoking, alcohol, lower fruit and vegetable intake, physical inactivity, obesity, high blood pressure, raised blood glucose and abnormal blood lipids.[6]

7. Recently, there were reports of high prevalence of these cardiovascular risk factors among industrial populations in various parts of India. Industrial units have captive population and therefore provide an opportunity to establish sentinel surveillance for cardiovascular risk factors.[11,13]

8. As many of the industrial units in organized sector have their own primary health care facilities, it provides an opportunity for routine screening and follow-up. In addition, worksites are considered to be a key channel for the delivery of interventions to prevent cardiovascular disease.[14]

9. Job strain defined as the confluence of high job demands and low job control, has been associated with increased CVD prevalence.[15] In Sweden, there are large differences in the incidence of myocardial infarction among occupational groups. Men aged <55yrs in high strain occupations showed an increase in the incidence of myocardial infarction compared to those in low strain occupations.[16] The remarkably high proportion of American executives who felt they were under high and increasing stress is consistent with their cholesterol levels, which were much higher than would be expected from the results of the National Health Survey.[17] It appeared that long working hours might increase systolic blood pressure in a field survey of 71 salesmen (22-60 years) in a machinery manufacturing company in Japan.[18]

In view of the above reasons it was decided to conduct this study among a group of sales executives in an urban industrial population.

REVIEW OF LITERATURE

HYPERTENSION

Hypertension is the commonest cardio-vascular disorder, posing a major public health challenge to societies in socio-economic and epidemiological transition. It is one of the major risk factor for cardio-vascular mortality which accounts for 20-50% of all deaths .[19] Hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease deaths in India.[20]

Prevalence

The prevalence of hypertension in India has been reported to range between 20–40% in urban adults and 12–17% among rural adults.[20] The number of people with hypertension is expected to increase from 118.2 million in 2000 to 213.5 million in 2025, with nearly equal numbers of men and women.[21]

Blood Pressure Definition and Classification

The arbitrary nature of the definition of hypertension has contributed to the variation in the definitions issued by various National and International authorities.

Following the announcement of the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT)[22] results at the end of 2002, the US hypertension guidelines were completely revised and reissued as the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and the Treatment of High Blood Pressure (JNC 7) in the Spring of 2003.

Hypertension is currently defined as a usual Blood Pressure of 140/90 mm Hg or higher, Blood Pressure levels for which the benefits of pharmacological treatment have been definitely established in randomized placebo controlled trials.[23]

Table 2- Classification of Blood Pressure for adults aged 18 years or older (JNC 7).[23]

Classification of Blood Pressure	**Systolic Blood Pressure (mm of Hg)**		**Diastolic Blood Pressure (mm of Hg)**
Normal	<120	And	<80
Prehypertension	120-139	Or	80-89
Stage 1 hypertension	140-159	Or	90-99
Stage 2 hypertension	≥160	Or	≥100

Unlike the current US hypertension guidelines (JNC 7), the WHO/ International Society of Hypertension guidelines are aimed at a global audience and are intended to serve as a template for the development of national, regional, and local guidelines. In the WHO/ISH statement, there is no "prehypertension" classification.

The World Health Organization (WHO)/ International Society of Hypertension (ISH) blood pressure classification includes 3 grades of hypertension (Table 2a).[24]

Table 2a - WHO/ISH Classification of Hypertension[24]

HYPERTENSION	**Systolic Blood Pressure (mm of Hg)**	**Diastolic Blood Pressure (mm of Hg)**
Grade 1	140-159	90-99
Grade 2	160-179	100-109
Grade 3	≥180	≥110

Accurate measurements of blood pressure are essential under standardized conditions for valid comparison between persons or groups over time. Three sources of errors have been identified in the recording of blood pressure.

1. Observer errors
2. Instrumental errors
3. Subject errors.

Etiology

Hypertension is divided into primary (essential) and secondary hypertension. In over 90% of patients with hypertension no specific cause can be identified. These patients are diagnosed as having primary hypertension or essential hypertension.

The small minority of patient in whom specific cause can be identified are diagnosed as having secondary hypertension. Prominent among these are

- Diseases of kidney
- Tumours of adrenal glands
- Congenital narrowing of the aorta
- Toxemia of pregnancy.

Cardiovascular Disease Risk

Subjects with hypertension are known to have a higher risk of **two-fold** developing **coronary artery disease**, **four times** higher risk of **congestive heart failure** and **seven time**s higher risk of **cerebrovascular disease** and stroke compared to normotensive subjects.[25] Even the high normal blood pressure (Systolic Blood Pressure

130 to 139 mm Hg, Diastolic Blood Pressure 85 to 89 mm Hg or both) augments risk of CVD twofold as compared with lower levels.[26]

As the population ages, the prevalence of hypertension will increase even further unless broad and effective preventive measures are implemented.[23] The relationship between Blood Pressure and risk of cardiovascular disease events is continuous, consistent, and independent of other risk factors. The higher the Blood Pressure, the greater the chance of myocardial infarction, heart failure, stroke and kidney disease. For individuals aged 40 to 70 years, each increment of 20 mm Hg in Systolic Blood Pressure (SBP) or 10 mm Hg in Diastolic Blood Pressure (DBP) increases the risk of CVD across the entire Blood Pressure range from 115/75 to 185/115 mm Hg.[27] The classification of prehypertension in JNC 7, recognizes this relationship and signals the need for increased education of health care professionals and the public to decrease Blood Pressure levels and prevent the development of hypertension in the general population.[28]

Hypertension prevention strategies are available to achieve this goal.

Lifestyle Modifications

Adoption of healthy lifestyles by all individuals is critical for the prevention of high Blood Pressure and an indispensable part of the management of those with hypertension. Major lifestyle modifications shown to lower Blood Pressure include weight reduction in those individuals who are overweight or obese; adoption of Dietary Approaches to Stop Hypertension eating plan, which is rich in potassium and calcium; dietary sodium reduction; physical activity; and moderation of alcohol consumption (Table 3).

Table 3. Lifestyle Modifications to Manage Hypertension*

Modification	Recommendation	Approximate Systolic BP Reduction, Range
Weight reduction	Maintain normal body weight (BMI, 18.5-24.9)	5-20 mm Hg/10-kg weight loss[23,24]
Adopt DASH eating plan	Consume a diet rich in fruits, vegetables, and low-fat dairy products with a reduced content of saturated and total fat	8-14 mm Hg[25,26]
Dietary sodium reduction	Reduce dietary sodium intake to no more than 100 mEq/L (2.4 g sodium or 6 g sodium chloride)	2-8 mm Hg[25-27]
Physical activity	Engage in regular aerobic physical activity such as brisk walking (at least 30 minutes per day, most days of the week)	4-9 mm Hg[28,29]
Moderation of alcohol consumption	Limit consumption to no more than 2 drinks per day (1 oz or 30 mL ethanol [eg, 24 oz beer, 10 oz wine, or 3 oz 80-proof whiskey]) in most men and no more than 1 drink per day in women and lighter-weight persons	2-4 mm Hg[30]

Abbreviations: BMI, body mass index calculated as weight in kilograms divided by the square of height in meters; BP, blood pressure; DASH, Dietary Approaches to Stop Hypertension.
*For overall cardiovascular risk reduction, stop smoking. The effects of implementing these modifications are dose and time dependent and could be higher for some individuals.

Source (23)

Pharmacologic Treatment

Excellent clinical trial outcome data prove that lowering Blood Pressure with several classes of drugs, including angiotensin converting enzyme inhibitors, angiotensin-receptor blockers, beta-blockers, calcium channel blockers, and thiazide-type diuretics, will all reduce the complications of hypertension.

Prevention and control of hypertension

The WHO recommended the following approaches for the prevention of hypertension.

1. Population approach which reduces the risk of developing high blood pressure in the population as a whole.
2. Individual approach which identifies the individuals with high blood pressure who are at increased risk of developing complications.

The combination of individual and population approach provides a comprehensive strategy for prevention and control of hypertension.

In population approach, the prevention of elevated Blood Pressure is principally linked to the elimination of modifiable risk factors that contribute to its rise and promotion of protective factors that help maintain blood pressure in the desirable range associated with low risk of complications.

This approach helps the individual to adopt a healthier lifestyle by keeping a normal body weight, eating less salt, consuming less alcohol, increasing physical activity and changes in behaviour such as cessation of tobacco smoking and improved diet. Encouraging change in behaviour in communities and individuals requires a collaborative effort between health professionals, policy makers, industry, media and other opinion formers as well as sustained campaign that targets all sections of the community and all ages.

The concept of the population approach is based on the fact that even a small reduction has the potential to produce not only a substantial reduction in the prevalence of hypertension, but a surprisingly large **decrease** in **cardiovascular risk**. The goal of the population approach is to shift the community distribution of blood pressure towards lower level or biological normality.

The individual approach focuses on the people at high risk. Family history of hypertension, tracking of blood pressure from childhood may be used to identify the individual at risk.

DIABETES MELLITUS

Definition

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both.[29]

The risk of CHD is 2-3 times higher in diabetics than non diabetics. CHD is responsible for 30 to 50 per cent of deaths in diabetics over 40 years in industrialized countries.[30]

Prevalence

The Indian subcontinent has a higher prevalence of diabetes mellitus than any other region in the world, and 2-3 times the reported prevalence in Western countries.[31] The prevalence of type-2 diabetes in urban Indian adults has been reported to have increased from less than 3% in 1970 to about 12% in 2000. The Indian Council of Medical Research (ICMR) estimates that the prevalence of diabetes is 3.8 per cent in rural areas, compared with 11.8 per cent in urban areas.[32]

Classification

Clinical classification of diabetes mellitus[30]

1. Diabetes mellitus
    i. Insulin-dependent diabetes mellitus (IDDM, Type 1)
    ii. Non-insulin dependent diabetes mellitus (NIDDM, Type 2)
    iii. Malnutrition-related diabetes mellitus (MRDM)
    iv. Other types (secondary to pancreatic hormonal, drug-induced, genetic and other abnormalities)
2. Impaired glucose tolerance (IGT)
3. Gestational diabetes mellitus (GDM)

Criteria for the diagnosis of diabetes mellitus[29]

1. Symptoms of diabetes plus casual plasma glucose concentration ≥200 mg/dl (11.1mmol/l). Casual is defined as any time of day without regard to time since last meal. The classic symptoms of diabetes include polyuria, polydipsia, and unexplained weight loss.

or

2. Fasting Plasma Glucose ≥126 mg/dl (7.0mmol/l). Fasting is defined as no caloric intake for at least 8hours.

or

3. 2-h postload glucose ≥200 mg/dl (11.1mmol/l) during an Oral Glucose Tolerance Test (OGTT). The test should be performed as described by WHO, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.

In the absence of unequivocal hyperglycemia, these criteria should be confirmed by repeat testing on a different day. The third measure (OGTT) is not recommended for routine clinical use.

Diabetes and CVD risk.

In men with diabetes, hypercholesterolemia, smoking, and hypertension predict coronary mortality risk, as well as mortality risk from all causes.[33] Even in the absence of the above risk factors diabetes alone results in increased relative risk of cardiovascular mortality.[34]

Diabetes is also strongly associated with classical risk factors- elevated levels of triglycerides and low-density lipoprotein cholesterol, with decreased levels of high-density lipoprotein cholesterol. There is also high prevalence of obesity and hypertension among diabetics.[35]

Prevention of Diabetes

Two strategies for primary prevention have been suggested.

- Population strategy
- High-risk strategy

Population strategy

The scope for primary prevention of NIDDM is limited on the basis of current knowledge and is not appropriate. However, the development of prevention programmes for NIDDM based on elimination of environmental risk factors is possible. There is pressing need for primordial prevention (prevention of emergence of risk factors in countries in which they have not yet appeared). The preventive measures comprise maintenance of normal body weight through adoption of healthy nutritional habits (adequate protein intake, a high intake of dietary fibre and avoidance of sweet foods) and physical exercise. These measures should be fully integrated into other community-based programmes for non-communicable diseases.

High-risk strategy

There is no special high-risk strategy for IDDM diabetes. At present, there is no practical justification for genetic counselling as a method of prevention.

Since NIDDM appears to be linked with sedentary lifestyle, over-nutrition and obesity, correction of these may reduce the risk of diabetes and its complication. Alcohol should be avoided. Those at risk should avoid diabetogenic drugs such as oral contraceptive pills. It is wise to reduce factors that promote atherosclerosis, e.g. smoking, high Blood Pressure, elevated cholesterol and high triglyceride levels. These programmes may most effectively be directed at target population groups.

DYSLIPIDEMIA

Definition

Dyslipidemia refers to the derangements of one or many of the lipoproteins; elevations of total cholesterol, Low-Density Lipoprotein (LDL) cholesterol and/or triglycerides, or low levels of High-Density Lipoprotein (HDL) cholesterol, while elevation of lipoproteins alone is labeled as 'hyperlipidemia'.[36]

Prevalence

The prevalence of hypercholesterolemia was 28% in urban subjects as compared to 22% in the rural subjects in a study among adult males in Rajasthan.[37] The ICMR surveillance project reported a prevalence of dyslipidemia (defined as a ratio of total to HDL cholesterol >4.5) of 37.5% among adults aged 15-64 yr, with an even higher prevalence of dyslipidemia (62%) among young male industrial workers.[38]

Classification of Total Cholesterol

Cholesterol is a fat-like substance, found in the blood stream and also in bodily organs and nerve fibers. Most cholesterol in the body is made by the liver from a wide variety of foods, but especially from saturated fats, such as those found in animal products.

Total cholesterol is the most widely used blood measure.[39]

Table 4 - Adult Treatment Panel III (ATP III) Classification of Total Cholesterol[40]

Total Cholesterol level	Classification
<200 mg/dl (5.2 mmol/L)	Desirable
200-239 mg/dl (5.2-6.2 mmol/L)	Borderline high
>240 mg/dl (6.2 mmol/L)	High

Total cholesterol is commonly divided into three major components based on the density of the particles: low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and Very Low-Density Lipoprotein cholesterol. Each of these fractions is associated with specific protein carrier molecules.

Low-density lipoprotein is the largest component of total cholesterol and is the atherogenic fraction. High-density lipoprotein is a smaller fraction and is inversely related to coronary heart disease. The very low density lipoprotein contains modest amounts of cholesterol, but is the main carrier for triglycerides. Triglycerides are the major entity by which fat is transported and stored in the body.

Etiology

A primary dyslipidemia (e.g. familial hypercholesterolemia) typically refers to a genetic defect in the lipid metabolism that causes abnormal lipid levels.

A secondary dyslipidemia may be due to a variety of reasons;

- environmental factors (diet rich in saturated fat or a sedentary lifestyle),
- diseases (type 2 diabetes, hypothyroidism, etc.), and
- medications (thiazide diuretics, progestins, anabolic steroids etc.).

Secondary dyslipidemias could be corrected or ameliorated by treating the underlying disorder.

Hypercholesterolemia and CVD

Globally, a substantial proportion of cardiovascular disease-one third of ischemic stroke and over half of Ischaemic Heart Disease is attributable to non-optimal cholesterol (defined as mean cholesterol >3.8 mmol/l).

- Data from prospective cohort studies have demonstrated a strong, continuous temporal association between cholesterol and Ischaemic Heart Disease
- The associations between age and cholesterol for males increased between the ages of 30 and 50 years, and then flattened before declining slightly in older age.
- The relative risk of Ischaemic Heart Disease increased with increasing cholesterol level, and the association was roughly linear when plotted on a log scale.
- Cholesterol is positively associated with ischemic stroke.[41]

A combination of hypertriglyceridemia, low levels of HDL-cholesterol and high levels of small dense low-density lipoprotein, termed as "atherogenic dyslipidemia', is particularly seen in Asian Indians.

A multitude of factors may contribute to dyslipidemia in Asian Indians.[36]

- Physical Activity: Asian Indians are more physically inactive as compared to many ethnic groups.
- Diet: Asian Indian diets rich in carbohydrate and low in ω-3 polyunsaturated fatty acids may exacerbate hypertriglyceridemia.
- Body Composition: Migrant Asian Indians and Asian Indians residing in urban India have body composition conducive to development of dyslipidemia.
- Genetic predisposition

OBESITY

Definition

Obesity may be defined as an abnormal growth of the adipose tissue due to an enlargement of fat cell size (hypertrophic obesity) or an increase in fat cell number (hyperplastic obesity) or a combination of both.[42]

Measurement and classification

Height and weight are the most simple and commonly used measures. A number of weight-for-height indices have been developed of which the **Body Mass Index** (BMI) is the most widely used. It is defined as the weight in kilograms divided by the square of the height in metres (kg/ m^2). Although it can sometimes misclassify total body fat content, BMI generally correlates highly with adiposity. Therefore BMI, which is easy to calculate, has been recommended as the measure of obesity for adults to be used in all studies.[43]

Table 5 - Classification of adults according to BMI (WHO Classification)

Classification	**BMI (kg/m^2)**	**Risk of co-morbidities**
Underweight	<18.5	Low (but increased risk of other clinical problems)
Normal range	18.5 – 24.9	Average
Overweight	≥ 25	
Preobese	25 – 29.9	Increased
Obese I	30 – 34.9	Moderate
Obese II	35 – 39.9	Severe
Obese III	≥ 40	Very Severe

It is not just the amount of fat but also its distribution that determines the risk associated with obesity. Abdominal or visceral fat (android obesity) is associated with the cardiovascular risk factors of the Metabolic Syndrome. These include Type 2 diabetes, impaired glucose tolerance, and hypertension and dyslipidaemia (high triglyceride, low LDL cholesterol). It is the mass of visceral adipose tissue which leads to these abnormalities.

Obesity and CVD risk

Increased mortality among the obese is evident for several life-threatening diseases including Type 2 diabetes, cardiovascular disease (relative risk 2-3, moderately increased risk), gallbladder disease, and certain types of cancers. Risks are also higher for some non-fatal conditions such as back pain, arthritis, infertility and, in many westernized countries, poor psychosocial functioning.

Long-term prospective data suggest that obesity may be an independent risk factor for CHD. The degree of obesity is related to the rate of development of CHD and even moderate overweight appears to increase risks of CHD. Weight loss accompanies improvements in blood pressure and cholesterol.

A proposal has been made to redefine the classification of obesity using BMI for Asian population (Table 6) as there are now evidence that the increased risks of co-morbidities with obesity occurs at a lower BMI in Asians.[44]

Table 6 - Classification of weight by BMI in adult Asians

Classification	BMI (kg/ m^2)	Risk of co-morbidities
Underweight	<18.5	Low (but increased risk of other clinical problems)
Normal range	18.5 – 22.9	Average
Overweight	≥23	
At risk	23 – 27.4	Increased Risk
Obese	≥27.5	High Risk

Prevalence

Many cross-sectional surveys, as well as the industrial surveillance project, recorded a high urban prevalence of central obesity and overweight (especially when the lower thresholds recommended by WHO for Asian people are used). Though the prevalence of obesity (BMI ≥ 30 kg/ m^2) is usually lower than that observed in the western population, the overweight category (BMI ≥ 25 kg/ m^2) includes almost a third to half the population in every survey.[45,46]

Prevention and control

Weight control is widely defined as approaches to maintaining body weight within the 'healthy' range of BMI of 18.5 to 24.9 kg/ m^2 throughout adulthood. It should also include prevention of weight gain of more than 5 kg in all people. In those who are already overweight, a reduction of 5-10 percent of body weight is recommended as an initial goal. The control of obesity centers around weight reduction. This can be achieved by dietary changes, increased physical activity as well as a reduction in smoking and alcohol consumption.

PHYSICAL ACTIVITY

Physical activity is defined as any body movement that results in the expenditure of energy. It includes sports and activities such as walking, cycling, playing, skating, cleaning the house, dancing or climbing the stairs. It is thus part of daily life.

There is international consensus on the value of regular moderate physical activity. This can be provided by *at least 30 minutes of physical activity daily*. The total of 30 minutes does not have to be performed in a single session; it can be accumulated throughout the day.[47]

Prevalence

As a result of economic changes and increased mechanization, the prevalence of physical inactivity is increasing in India, particularly in urban areas, to levels comparable with the West.[48] In the INTERHEART study, the prevalence of leisure time physical activity was substantially lower among South Asians (6.1%) compared with the rest of the world (21.6%).[49]

Physical activity and CVD risk

Regular physical activity reduces the risk of obesity, blood lipid abnormalities, hypertension, and non-insulin dependent diabetes mellitus[50], and has been shown to reduce substantially the risk of CHD. Conversely, measures of sedentary lifestyles or physical inactivity have been associated with a 1.5 to 2.4 fold elevation in CHD risk[51] A recent hospital-based case control study from two urban centers in India suggested that daily moderate intensity physical activity (e.g., the equivalent of briskly walking 35- 40 min per day) is associated with a 55 per cent lower risk for CHD[52].

Benefits to health of physical activity[47]

Physical activity has the following benefits:

- a 50% reduction in the risk of developing coronary heart disease, adult diabetes or obesity;
- a 30% reduction in the risk of developing hypertension;
- a decline in blood pressure in hypertensive people;
- maintenance of bone mass and thus protection against osteoporosis;
- improved coordination, mobility, strength and endurance; and
- raised self-esteem, reduced levels of mild to moderate hypertension and overall psychological wellbeing.

SMOKING

Prevalence

In 2002, a national survey of tobacco use reported that the Indian subcontinent, second only to China in both the production and consumption of tobacco products, had an alarming rate of current tobacco use of 56 per cent among Indian men aged 12-60 yr.[53] Data from the Global Youth Tobacco Survey shows that the Proportion currently using any tobacco product among 13 to 15 years old is 17.3 % (males) and 9.7% (females) and the Proportion currently smoking cigarettes 5.9 % (males) and 1.8% (females).

Smoking and Cardiovascular risk

Cigarette smokers have higher death rates from coronary heart disease than nonsmokers. This relationship is stronger for men than women. Cigarette smoking

markedly increases an individual's susceptibility to earlier death from coronary heart disease. Cigarette smoking, hypertension and elevated serum cholesterol are major risk factors contributing to the development of coronary heart disease. Cigarette smoking acts both independently and conjointly with these other factors to increase the risk of developing coronary heart disease. Cigarette smoking may contribute both to the development of coronary heart disease and to the exacerbation of pre-existing coronary heart disease; both nicotine and carbon monoxide are thought to contribute to these abnormal processes. Cigarette smoking is associated with a significant increase in atherosclerosis of the aorta and coronary arteries. Cessation of smoking is associated with a decreased risk of death from coronary heart disease.[54] The degree of developing coronary heart disease is directly related to the number of cigarettes smoked per day[55]
New data reaffirm the already well documented causal association of smoking with the risk for CHD. Compared with lifetime nonsmokers, the relative risk in smokers rises with the number of cigarettes smoked and falls after cessation. The type of cigarette smoked has little influence on CHD risk. Products with lower yields of tar and nicotine, as measured by a smoking machine, have not been found to reduce CHD risk substantially and they are not a lower-risk alternative for smokers who cannot quit.[56]

Little data have existed regarding the association between the use of other forms of tobacco and the risk of CVD; however, a recent analysis of data from the INTERHEART case-control study of risk factors for acute myocardial infarction has documented that there is an increased risk of myocardial infarction associated with all forms of smoked and smokeless tobacco.[57]

Prevention Strategy

The tobacco epidemic is preventable. In May 2003, the WHO World Health Assembly unanimously adopted the WHO **Framework Convention on Tobacco Control** (FCTC), one of the United Nations' most widely embraced treaties – and the world's first against tobacco – in order to galvanize action at the global and country level against the tobacco epidemic.

To expand the fight against the tobacco epidemic, WHO has introduced the **MPOWER**[58] package of six proven policies:

- Monitor tobacco use and prevention policies,
- Protect people from tobacco smoke,
- Offer help to quit tobacco use,
- Warn about the dangers of tobacco,
- Enforce bans on tobacco advertising, promotion and sponsorship, and
- Raise taxes on tobacco.

The MPOWER[58] policy package can reverse the tobacco epidemic and prevent millions of tobacco-related deaths.

Legal provisions for Tobacco control in India and Tamil Nadu

Cigarettes and Other tobacco products (Prohibition of Advertisement and Regulation of Trade and Commerce, Production, Supply and Distribution) Act, 2003. Four Key Areas :

- Prohibition of smoking in a public place – section 4
- Prohibition of advertisement of cigarettes and other tobacco products – section 5

- Prohibition on sale of cigarettes or other tobacco products to and by minors and in a particular area – section 6
- Specified health warning labels on all tobacco products including pictorial warnings– sections 7,8,9,10.

The Tamil Nadu Prohibition of Smoking And Spitting Rules 2003.

ALCOHOL

High alcohol intake defined as 75 g or more per day is an independent risk factor for CHD, hypertension and all cardiovascular diseases.[59]

Evidence that regular light-to-moderate drinking has a beneficial effect on the cardiovascular system comes from physiological and individual-level epidemiological studies. How-ever, aggregate-level, time-series analyses have failed to confirm this effect.

Binge drinkers (i.e., women who consumed five or more drinks on an occasion, or men who consumed nine or more drinks on an occasion) had higher risks for major coronary events than abstainers, even when over-all volume of drinking was low.

Specifically, heavy drinking occasions have been shown to increase low density lipoproteins, which have been linked to negative cardiovascular outcomes.

In addition, irregular heavy drinking is associated with increased risk for the formation of blood clots within blood vessels (i.e., thrombosis), which occurs at the end of a heavy drinking occasion.

Finally, irregular heavy drinkers seem predisposed to structural (i.e., histological) changes in the heart muscle and the adjacent impulse-conducting system, which regulate the threshold for ventricular fibrillation.[60]

In the face of good evidence that shows a relationship between consumption of alcoholic beverages and various types of cardiovascular harm, in addition to the considerable doubt about the benefits with respect to ischaemic heart disease, alcohol should be treated as a risk factor in the aetiology of cardiovascular diseases.[61]

SALES EXECUTIVE

A person employed to represent a business and to sell its merchandise.[62]

Job description of a sales executive[63]

Sales executives maximise the sales of a company's goods or services in many different settings. Their role helps to ensure the commercial success of a diverse range of companies.

Sales executives are responsible for increasing and developing sales in areas including:

- fast moving consumer goods (such as food and stationery);
- consumer durables (clothes, domestic equipment and toys);
- industrial supplies;
- IT, software and media;
- services (such as print and financial services).

The requirement to meet and exceed targets means that the role is demanding and involves working under frequent pressure. Travel within a working day, absence from home at night and overseas work or travel, are all common. The constant travel, sometimes with overnight stays or longer business trips, can affect social/home life.

The work environment has most often been used to study chronic stress as it leads to coronary artery disease.[64] The most commonly used demand/control model hypothesis predicts that the most adverse reactions of psychological strain occur when the psychological demands are high and the worker's decision latitude is low: job strain. According to Robert Karasek, two scales, decision latitude and psychological demands are used to measure the high demand/low control model of job strain development.[65] When the two job characteristic scales-decision latitude and psychological demands-are arrayed as a four-quadrant diagram, we get:

- Active job quadrant (upper right) with high demand and high control, has high-prestige occupations: public officials, physicians, engineers, nurses, and managers of all kinds.
- Passive job quadrant (lower left), with low demands and low control, has clerical workers such as billing clerks, and low-status service personnel such as janitors.
- High-strain quadrant (lower right), with high demands and low control, has machine-paced operatives such as assemblers, cutting operatives, freight handlers, salespersons as well as other low-status service operatives such as waiters.
- Low-strain self-paced occupations (upper left) often involve significant training and self-pacing, such as repairmen, linemen, and natural scientists.

Organized sector

Manufacturing sector plays a vital role in Indian economy. It contributes almost 25% of GDP in the Indian economy. Manufacturing sector is further subdivided into 2 parts-organized sector and unorganized sector. Organized sector means the units registered under Section 2m of Factories Act, 1948 and unorganized sector means the rest.[66]

Definition of Industry

As per Section 2(j) of Industrial Disputes Act, 1947 "Industry" means any systematic activity carried on by co-operation between an employer and his workmen (whether such workmen are employed by such employer directly or by or through any agency, including a contractor) for the production, supply or distribution of goods or services with a view to satisfy human wants or wishes (not being wants or wishes which are merely spiritual or religious in nature).

A new initiative for a healthy nation

National Programme for Prevention and Control of Diabetes, Cardiovascular Diseases and Stroke (NPDCS)[67]

Pilot phase of the National Programme for Prevention and Control of Diabetes, Cardiovascular Diseases and Stroke (NPDCS) was launched on 4th Jan 2008 by Deputy Chairman, Planning Commission in the august presence of Honorable Minister for Health &Family Welfare and Honorable Minister of State.

States and Districts covered for Pilot Phase

One district in each of the following states covered for pilot Phase

Table 7 - States and Districts selected for the pilot phase

State	District
Assam	Kamrup
Punjab	Jalandhar
Rajasthan	Bhilwara
Karnataka	Shimoga
Tamilnadu	Kancheepuram
Kerala	Thiruvanathapuram
Andhra Pradesh	Nellore(tentative)

Objectives of the pilot phase

1. To assess the prevalence of risk factors for Non- Communicable Diseases
2. Risk reduction for prevention of NCDs (Diabetes, CVD and Stroke)
3. Early diagnosis and appropriate management of Diabetes, Cardiovascular Diseases and Stroke

Strategies

a. Surveillance of risk factors of Non-Communicable Diseases.

b. Health Promotion for the General Population.

c. Disease Prevention for the High Risk groups.

a. Assessment of prevalence of NCD risk factors

To study the distribution of risk factors for Non-Communicable Diseases especially Diabetes, CVD and Stroke-through repeated surveys in the population will include

1. Demographic and socio-economic characteristics
2. Tobacco/Alcohol use
3. Physical activity
4. Dietary patterns
5. BMI and waist circumference measurements
6. Blood pressure
7. Blood glucose and Lipids

b. Health Promotion

1. Enabling environments

Through an effective communication & community mobilization in

i. Schools

ii. Communities (rural and urban)

iii. Workplaces

2. Implementation through

Non Governmental Organizations, School Health Committees, Ward Committees, Accredited social health activists ASHAs, Self Help Groups and Employees Unions

Key issues to be addressed

1. Healthy food habits
2. Control of Overweight and Obesity
3. Regular physical activity
4. Avoidance of tobacco and
5. Stress management
6. Control of Blood Pressure, Blood Sugar and Blood Cholesterol/Lipids.

c. Disease prevention for the High Risk

will be achieved through

i. Reorienting the Public Health Delivery system.

ii. Setting up of special clinics.

iii. Harnessing the Private Sector.

iv. Specific interventions at tertiary level.

Expected outcomes for the pilot phase

- Population distribution of NCD risk factors made available
- Awareness generated on HEALTHY LIFE STYLES
- Health promotion at School , Community & Work places
- Health system capacity building for prevention and control of NCDs
- Methodology and approaches finalized for Nationwide implementation.

METHODOLOGY

BACK GROUND INFORMATION

The study was conducted in the corporate office of a Fast Moving Consumer Goods Company in South Chennai. The office was selected based on the feasibility of long term follow up of the cohort with limited resources. An in-house functional medical center with a physician, medical aide and a records section was also present. There were nearly 1000 employees working in the office in various capacities. A total of 676 persons were employed as sales executives. In this particular organization, the sales employees were required to visit the depots and retail stores and check for supply and availability of stock, in the areas of their jurisdiction which covered various areas in the city of Chennai. This required travel approximately 50-70 kilometers per day, irregular diet habits (eating outside and improper timings) and irregular working hours.

STUDY DESIGN

This study was done as a cross-sectional descriptive study of all sales executives in the 20–59 years age group.

STUDY PERIOD

The study was conducted during the period between July 2008 to September 2008.

STUDY POPULATION

A list of all potential participants was obtained from the human resources department. A total of 676 persons were employed as sales executives. All of them were invited to participate in the study. Of the 676 individuals, 608 (89.94% of total, 5 women

and 603 men) agreed to participate in the study. Here the results of the men are reported, as the number of women who participated in the study was small 5 (0.82%).

SAMPLE SIZE

All the eligible employees were included in the study; however for estimation of the sample size, the following calculation was used.

Formula

$$\mathbf{n = \frac{Z\alpha^2\, pq}{d^2}}$$

where $Z\alpha^2 = 1.96$ for 95% confidence interval.

Here the sample size was arrived at with the prevalence of major CVD risk factor hypertension 27.2% as reported in a study conducted among an urban industrial population in South India.[68]

n = number of samples to be studied

p = 27.2%

q = 72.8%

d = 15% of p

= 15% of 27.2 = 4.08

Substituting values in the formula

$$n = \frac{Z\alpha^2\, pq}{d^2}$$

We get,

$$n = \frac{1.96\times1.96\times27.2\times72.8}{4.08\times4.08} = 456.97$$

Sample size = 457

SELECTION CRITERIA

Inclusion criteria: Adults who are willing to participate in the study.

Exclusion criteria: Those who were not willing participate in the study.

DATA COLLECTION

Approval for conducting the study was obtained from the management. Participation in the study was voluntary and all the measurements were carried out in the medical unit located within the industry under the supervision of the in house physician. Informed oral consent was obtained from each participant after explaining about the nature of the study. Data on socio-demographic aspects, tobacco consumption, alcohol consumption, physical activity, anthropometric measurements, blood pressure measurements and blood samples for biochemical measurements were collected from all the study subjects. All the study subjects were individually counselled on methods to reduce risk factors. Newly detected patients with hypertension, diabetes, and hypercholesterolemia were referred to the in house physician for further management. At the end of the study, a copy of the report of the study was submitted to the management.

STUDY METHOD

The three components of the study were.

(1) Questionnaire based survey for behavioural risk factors,

(2) Anthropometric measurements

(3) Blood Pressure and Biochemical measurements.

Questionnaire for demographic and behavioural risk factors

A simple semi-structured questionnaire consisting of objective type questions was developed and modified by pilot testing and on expert advice.

The questionnaire was used to collect data on demographic and behavioural risk factors. The first part sought information on the socio-demographic aspects like age, sex, occupation, education, monthly income and marital status.

The second part sought self reported information on lifestyle-related factors such as physical activity, smoking and alcohol consumption; and any history related to previous diagnosis and treatment for hypertension and diabetes.

Questions on tobacco included data on self reported frequency and quantity of tobacco consumption. Individuals were classified as non-smoker or smoker (only one person consumed smokeless tobacco and all the others smoked cigarettes).

Self reported alcohol intake data was collected and subjects were classified as consumer and not a consumer.

Anthropometric measurements

Weight was measured in the upright position with the help of an adult portable-weighing machine (bathroom scale). The same machine was used throughout the study.

The scale was adjusted to zero reading before weighing. Accuracy of the scale was checked every week with standard weight. Weighing was done with minimum dressing and without shoes.

Height was measured without shoes using calibrated stadiometer.

Body mass index (BMI) was calculated using the Quetelet's index.

$$\text{B.M.I.} = \frac{\text{Weight in kg}}{\text{Height in m}^2}$$

Measurement of Blood Pressure

Blood pressure was measured from the right arm after the subject had been sitting quietly with the back supported for at least five minutes and arm supported at the level of the heart.

1. No caffeine during the hour preceding the reading.
2. No smoking during the fifteen minutes preceding the reading.
3. No exertion of any kind preceding the measurements.[69]

The gold standard for routine blood pressure measurement is a Standard mercury column sphygmomanometer with cuff size 13cms x 35cms. The bladder should encircle and cover 2/3rd of the length of the arm. The lower edge of the cuff should be about 2.5cms above the anterior cubital fossa. The manometer should be placed on a horizontal surface at the level of the heart.

The blood pressure was recorded with the same sphygmomanometer and by the same observer. '0' reading was ensured before recording the blood pressure each time. The cuff pressure was inflated to 30mmHg above the level at which the radial pulse disappeared, then deflated slowly at the rate of 2mm/second and the reading recorded to the nearest 2mm. The first and the fifth Korotkoff sounds were taken as indicative of the systolic and diastolic blood pressure respectively.

Two readings were made at five minutes interval as per the WHO recommendation.

When high blood pressure greater than or equal to 140/90mmHg was noted a third reading was made after thirty minutes. Lowest of the three was recorded.

Biochemical measurements

Five ml of blood was collected from ante-cubital vein in two test tubes. Blood sample for plasma glucose was collected in the test tube containing heparin sodium fluoride. Plasma glucose and total cholesterol were measured using an auto-analyzer. The GOD – POD (glucose oxidase – peroxidase) method and CHOD – PAP (cholesterol oxidase / p – aminophenazone) method were used for measuring plasma glucose and serum cholesterol respectively at an accredited laboratory. Re-analysis of 10% of samples selected randomly was done at the Institute of Biochemistry, Madras Medical College as a quality assurance measure.

Statistical analysis

Data was entered in EXCEL-2003 and analyzed using Epi-info2002 version-6 computer package (developed by Center for Disease Control and Prevention (CDC), U.S.A. and WHO, Geneva). ANOVA test was used between mean values of CVD risk factors and age groups. Continuous data are summarized as means and standard deviations and categorical data as proportions. Chi-square test was used for categorical variables and P-value < 0.05 was considered statistically significant. Odds ratios were calculated between other risk factors and hypertension.

DEFINITIONS

Smoking

Current smoker: Smoked at least one cigarette during the last 30 days.[70]

Alcohol

Current drinkers: those who consumed at least one alcoholic beverage in the past 30 days. A drink was defined as a 355ml beer, 148ml wine, and 37ml liquor.[71]

Current regular use: Alcohol intake for more than three times (average) a week.

Current occasional use: Alcohol intake for more than three times a month, but less than three times a week.[11]

BMI classification: Subjects were classified using WHO classification[43] and classification recently recommended for Asians.[44]

Hypertension: Defined as systolic blood pressure (SBP) of ≥140 mmHg or diastolic blood pressure (DBP) ≥90 mmHg as per WHO criteria[24] or history of previously known disease and pre-hypertension was defined as SBP 120-139 mmHg or DBP 80-89 mmHg.[23]

Type 2 Diabetes mellitus: Diagnosed either by history of previously known disease or fasting plasma glucose of ≥126 mg/dl and impaired glucose tolerance was defined as plasma glucose 101 – 125 mg/dl.[29]

Hypercholesterolemia: Defined as total cholesterol level ≥ 200 mg% according to USA - adult treatment panel (ATP) III guidelines.[40]

RESULTS AND DISCUSSION

ORGANIZATION OF DATA

SECTION – A: DEMOGRAPHIC CHARACTERISTICS

SECTION – B: PHYSIOLOGIC BASELINE DATA

SECTION – C: PREVALENCE OF RISK FACTORS

SECTION – D: EFFECT OF AGE ON CVD RISK FACTORS

SECTION – E: EFFECT OF ADIPOSITY ON CVD RISK FACTORS

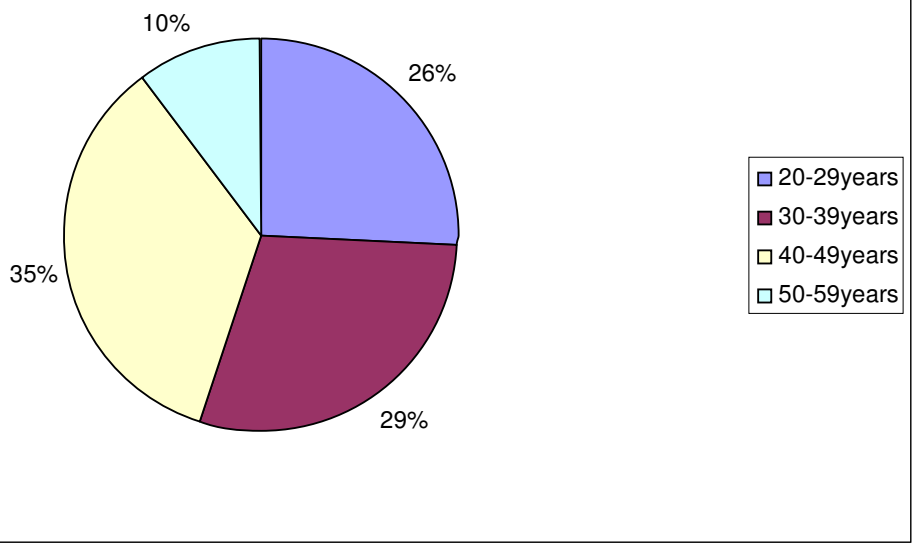
SECTION – F: ASSOCIATION BETWEEN HYPERTENSION AND OTHER RISK FACTORS

SECTION – A. DEMOGRAPHIC CHARACTERISTICS

The study was conducted in a corporate office in south Chennai. Of the 676 sales executives, 608 individuals (89.94% of total, 5 women and 603 men) agreed to participate in the study. Here the results of the men are reported, as the number of women who were eligible and participated in the study was small 5 (0.82%).

The study population included 603 males aged 22-59 years. The blood samples were collected for all subjects. Mean age was 37.68 ±9.19years. Nearly one third 209(34.7%) of the participants were in the 40–49 years' age group. All had graduate or post-graduate level education. All were semi-professionals belonging to the upper middle class, average family income was Rs.11,500 per month (8000-15000). Among the study subjects 537(89.1%) were married and 66(10.9%) were unmarried, 107(17.7%) were vegetarians and 496(82.3%) were mixed-diet consumers.

FIGURE-1: AGE DISTRIBUTION



SECTION – B. PHYSIOLOGIC BASELINE DATA

The mean values of various clinical, laboratory and anthropometric measurements are given in Table 8.

Table – 8: Mean And Standard Deviation For Various Cardiovascular Disease Risk Factors In The Study Population

S.No.	Variables	Mean	Standard Deviation	Range
1.	Age(years)	37.68	9.19	22-59
2.	Height(cm)	172.03	6.32	149-188
3.	Weight(kg)	75.81	11.05	45-121
4.	Body Mass Index(kg/m^2)	25.61	3.42	15.55-37.74
5.	Systolic Blood Pressure(mmHg)	130.25	14.62	100-200
6.	Diastolic Blood Pressure(mmHg)	84.15	9.27	60-130
7.	Fasting Blood Sugar(mg/dl)	100.96	41.69	70-351
8.	Total Serum Cholesterol(mg/dl)	203.00	38.34	54-368

Anthropometric measurements

The results showed a mean BMI of 25.61 kg/m^2 which is higher than the Normal range (18.5 – 24.9 kg/m^2). It is in the Pre-obese range (25 – 29.9 kg/m^2).

Clinical and biochemical measurements

Blood pressure

Mean systolic blood pressure (SBP) was 130.25mmHg and mean diastolic blood pressure (DBP) was 84.15mmHg.

The normal SBP is <120mmHg and DBP is <80mmHg. So the mean Blood Pressure values for the study population are higher than normal they are in the pre-hypertensive range.

Biochemical measurements

Mean Fasting Blood Sugar was 100.96mg/dl.

The mean Fasting Blood Sugar value for the study population is higher than normal. It is in the pre-diabetic range (100-125mg/dl).

Mean Total Serum Cholesterol was 203.00mg/dl

The mean Total Serum Cholesterol value for the study population is higher than desirable. It is in the Borderline high range (200-239 mg/dl)

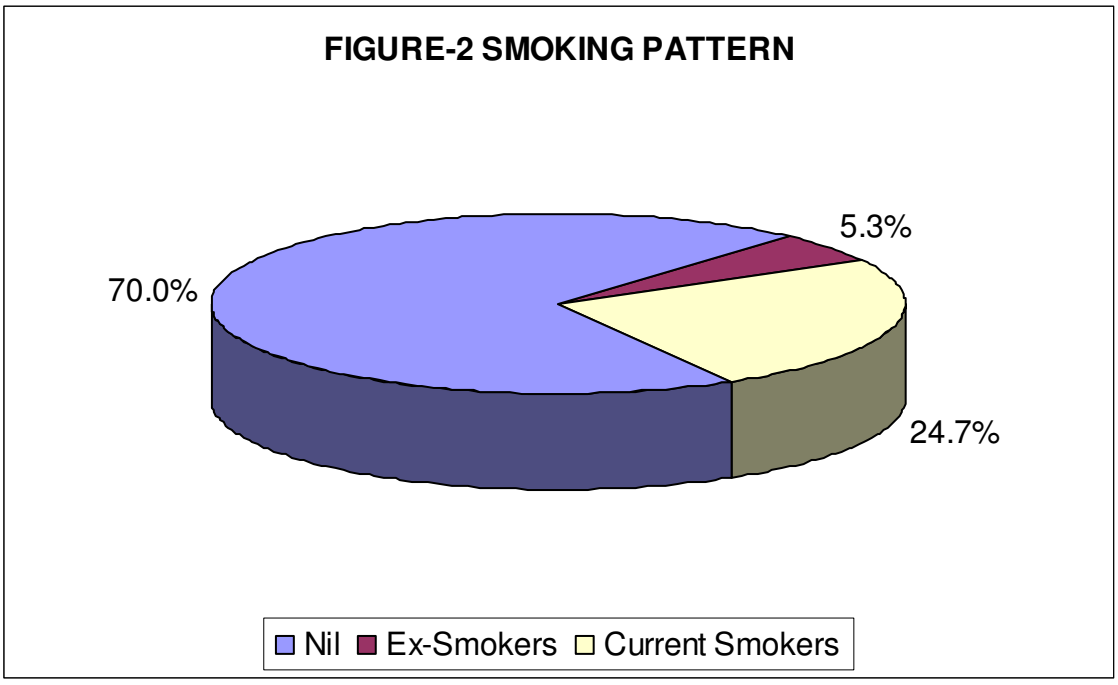
SECTION – C. PREVALENCE OF RISK FACTORS

BEHAVIOURAL RISK FACTORS

Community-based cross-sectional surveys and other studies carried out in the past few decades have revealed a high and increasing prevalence of CVD and its risk factors in Indians residing, in India[72,73,74,75] as well as in other countries.[76,77]

SMOKING

The number of study subjects who were smokers (ever smoked) was 181(30.0%, 95% Confidence Interval 26.4% to 33.9%), current smokers were 149 (24.7%) and ex-smokers 32(5.3%) as seen in (Fig-2).



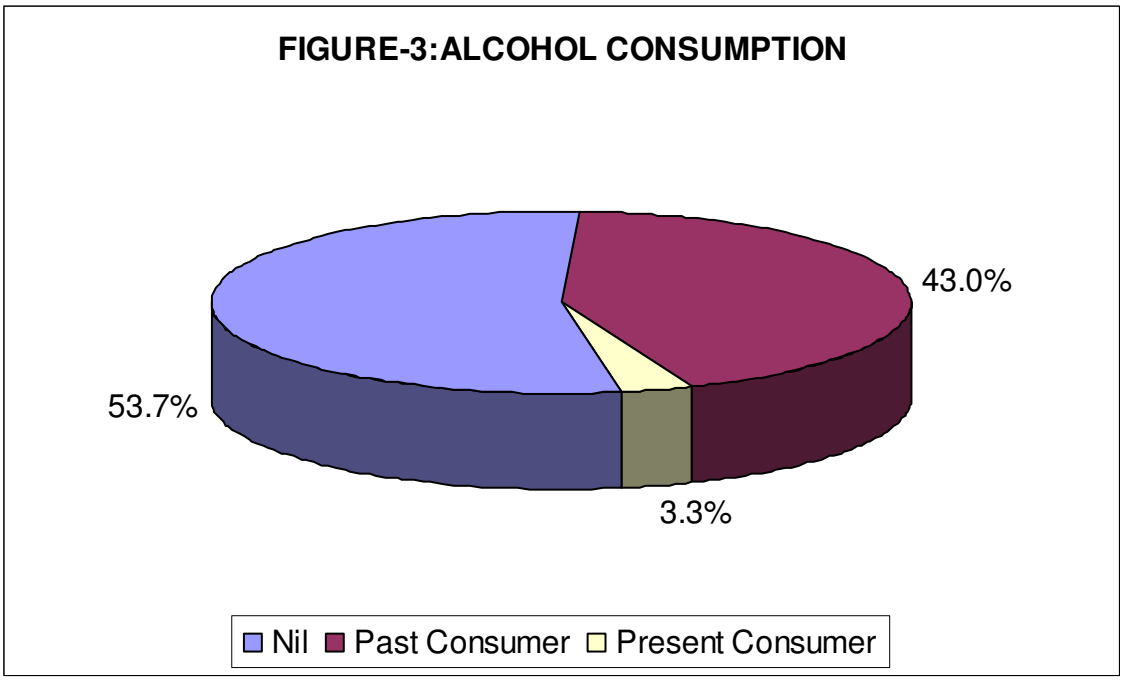
Similar results were found in other studies conducted in industrial settings. Kaur et al., [78] reported a prevalence of 33.3% smoking in their study, Mehan et al. ,[79] reported that smoking was prevalent in 31.4% study subjects in their profile of NCD risk factors in

an industry in Gujarat and Gupta et al.,[80] reported a prevalence 36.5% among the urban population of Jaipur.

ALCOHOL CONSUMPTION

Alcohol consumption was prevalent in 279(46.3%, 95% Confidence Interval 42.2% to 50.3%) subjects (ever consumed). Of the study subjects 3.3% were past consumers and 43.0% were present consumers (Fig-3).

FIGURE-3:ALCOHOL CONSUMPTION



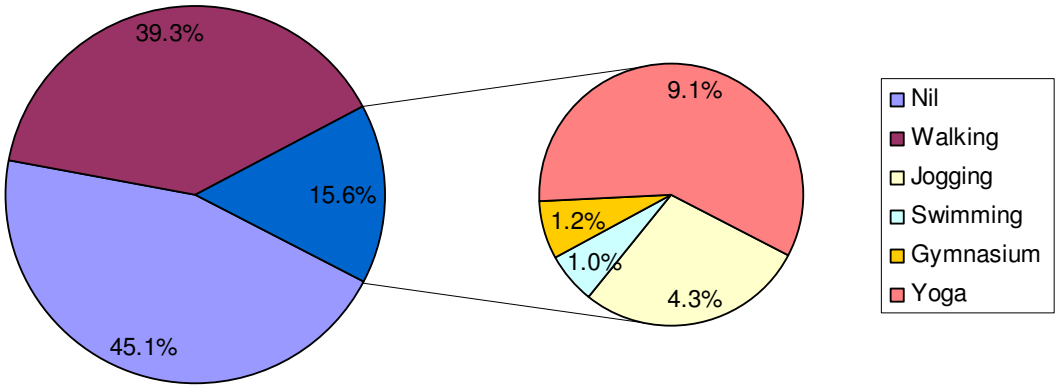
Kaur et al., reported a prevalence of 34.8% in their study done to determine the prevalence of cardiovascular risk factors in two industrial units in Chennai, India. Studies in northern India found the 1 year prevalence of alcohol use to be between 25 and 40%.[81] In southern India, the prevalence of current alcohol use varies between 33 and 50%, with a higher prevalence among the lesser educated and the poor.[82] Mohan et al., [83] (2001) conducted a survey in three districts (central, north and north-east India), they reported a prevalence of current alcohol use of 20–38% in males and of 10% among females.

PHYSICAL ACTIVITY

Apart from daily activities 331(54%, 95% Confidence Interval 50.8% to 58.9%) of the participants took part in some kind of physical activity. The various types of physical activities performed are shown in Fig.-4.

Physical inactivity was prevalent among 272 (45.1%, 95% Confidence Interval 41.1% to 49.2%) of the study subjects.

FIGURE-4: PHYSICAL ACTIVITY



Gupta R. et al,[84] reported the prevalence of physical inactivity (defined as-walk less than 14.5km per week, climbs less than 20 flights of stairs per week, or no moderate physical activity (300kcal/day) on 5 days per week) 85% (81% in males and 92% in females). Kaur et al., reported that based on the physical activity levels, 243(10.7%) in their industrial population were sedentary and 675 (29.8%) had heavy physical activity levels.

ANTHROPOMETRIC MEASUREMENTS

BODY MASS INDEX

Prevalence of overweight (BMI 25.0 kg/m2-29.9 kg/m^2) and obesity (BMI ≥ 30.0 kg/m^2) using WHO definition was 44.9%and 10.1% respectively. The overall prevalence of over weight (BMI >25 kg/m^2) was 55.1%, 95% Confidence Interval 51.0% to 59.1%.

Table – 9: Frequency And Percentage Distribution Of Body Mass Index (WHO Definition)

S.No.	Classification	BMI (kg/m^2)	Frequency	Percent
1.	Underweight	<18.5	15	2.5%
2	Normal range	18.5 – 24.9	256	42.5%
3.	Pre-obese	25 – 29.9	271	44.9%
4.	Obese	30 – 40	61	10.1%

As per WHO recommendations for defining risk thresholds among Asians, 52.9% were in increased risk (BMI 23.0 kg/ m^2-27.4 kg/m^2) and 26.5% were in high-risk (BMI ≥ 27.5 kg/m^2) category, the prevalence of overweight based on the WHO recommendations for Asians (BMI >23 kg/m^2) was 79.4%.(Table10)

Table – 10: Frequency And Percentage Distribution Of Body Mass Index

(WHO Recommendation for Asians)

S.No.	Classification	BMI (kg/m^2)	Frequency	Percent
1.	Underweight	<18.5	15	2.5%
2	Normal range	18.5 – 22.9	109	18.1%
3.	Pre-obese	23 – 27.4	319	52.9%
4.	Obese	27.5 – 40	160	26.5%

Mean BMI and prevalence of overweight and high-risk BMI in this population was higher as compared to other industrial settings. This could be due to better socio-economic status and more urbanized study population.

CLINICAL AND BIOCHEMICAL MEASUREMENTS

HYPERTENSION

Based on history of previously known disease, only 9.1% were aware of their blood pressure status. (Table-11)

Table – 11: Past History Of Hypertension

S.No.	History of Hypertension	Frequency	Percent
1.	Not a Known Hypertensive	548	90.9%
2	Known Hypertensive on Treatment	55	9.1%
3.	Total	603	100.0%

Table – 12: Frequency And Percentage Distribution Of Hypertension

S.No	Hypertension	Frequency	Percent
1.	Normal	54	8.9%
2.	Pre-hypertension	247	41.0%
3.	Self Reported	55	9.1%
4.	Newly Detected	247	41.0%
5.	Total	603	100.0%

In this study pre-hypertension was present in 247(41.0%) and hypertension was prevalent in 302 (50.1%, 95% Confidence Interval, 46.0% to 54.1%) subjects respectively. Among the hypertensives, 247 (81.78%) were newly detected (Table-12).

Table – 13: Classification Of Blood Pressure Levels (Combined) n=603

S.No	Category	(mm of Hg)		Frequency		Total (Percent)
		SBP	DBP	Newly detected	Known hypertensive	
1	Normal B.P.	<120	<80	54	0	54(9.0%)
2	Pre-hypertension	120-139	80-89	247	8	255(42.3%)
3	Grade 1 hypertension	140-159	90-99	195	25	225(36.5%)
4	Grade 2 hypertension	160-179	100-109	48	18	66(10.9%)
5	Grade 3 hypertension	≥180	≥110	4	4	8(1.3%)

Among the hypertensives in the study population, 36.5%were grade 1 or mild degree hypertension, 10.9% were grade 2 or moderate degree of hypertension and 1.3% were grade 3 or severe hypertension. Among known hypertensives only 8(14.54%) had blood pressure under control (SBP <140 mmHg and DBP<90 mmHg).

Chokalingam A et al.,[85] found that pre-hypertension was prevalent in 47.4% of adults, and another 34.7% had hypertension in a cross-sectional survey among 2,007 adults in Chennai in July 2003. Prabhakaran et al.,[86] reported a prevalence of pre-hypertension of 40% and hypertension 20% among men in a large industry of northern India. Kaur et al., reported findings of pre-hypertension 39.8% and hypertension 27.2% in an industrial population in south India.

The prevalence of hypertension among the subjects in this study was higher than that observed in other studies. This could be due to the unique work profile of the study population. Hazarika et al.,[87] in their study on Hypertension and its risk factors in tea garden workers of Assam also found a higher prevalence of hypertension (60.8%).

Consistent with other industrial studies, awareness and control of hypertension was low. Hypertension remains undetected and uncontrolled even in organized sector industries with medical facilities.

DIABETES

Based on history of previously known disease**,** self reported prevalence of diabetes was 9.6%.

Table – 14: Past History Of Diabetes

S.No.	History of Diabetes	Frequency	Percent
1.	Not a Known Diabetic	545	90.4%
2	Known Diabetic on Treatment	58	9.6%
3.	Total	603	100.0%

Impaired fasting glucose was prevalent in 85(14.1%) and Diabetes Mellitus was prevalent in 86(14.3%, 95% Confidence Interval 11.6% to 17.4%) subjects respectively. Among the diabetics, 28(32.6%) were newly detected. Among known diabetics, 29 (50.0%) had blood glucose under control (<126 mg/dl).

Table – 15: Frequency And Percentage Distribution Of Diabetes

S.No.	Diabetes	Frequency	Percent
1.	Normal	432	71.6%
2.	Impaired Fasting Glucose	85	14.1%
3.	Self Reported	58	9.6%
4.	Newly Detected	28	4.7%
5.	Total	603	100.0%

The prevalence of diabetes in this study was consistent with the recent Indian studies. Prabhakaran et al., observed that the prevalence of impaired fasting glucose30.7% and Diabetes Mellitus was 15%. Kaur et al.,[78] observed that the prevalence of impaired fasting glucose 16.1% and Diabetes Mellitus was 16.3%. In the multi-centric study done in industrial settings in India the prevalence of Diabetes Mellitus was found to be 10.1% and Gupta et al., reported a prevalence of 13.1% Diabetes Mellitus among men.

HYPERCHOLESTEROLEMIA

Hypercholesterolemia (total cholesterol ≥200 mg/dl) was observed in 308(51.1%, 95% Confidence Interval 47.0% to 55.1%) subjects.

Table – 16: Frequency And Percentage Distribution Of Hypercholesterolemia

S.No.	Serum Cholesterol	Frequency	Percent
1.	Normal(<200mg/dl)	295	48.9%
2.	Hypercholesterolemia(≥200mg/dl)	308	51.1%
3.	Total	603	100.0%

Kaur et al., found the prevalence of Hypercholesterolemia to be 30.3% in their study done to determine the prevalence of cardiovascular risk factors in two industrial units in Chennai, India, Prabhakaran et al., observed that the prevalence of Hypercholesterolemia was 30.1%, Gupta et al.,[80] reported a prevalence of 28.5% among the urban population of Jaipur.

The high prevalence of Hypercholesterolemia in this study population may be due to better socio-economic status and their dietary habits.

SECTION – D. EFFECT OF AGE ON CVD RISK FACTORS

Table – 17: Age Distribution of CVD Risk Factors **n = 603**

Cardio-vascular disease risk factor		20-29 years n=156		30-39 years n=176		40-49 years n=209		50-59 years n=62		Chi-square
		No.	%	No.	%	No.	%	No.	%	
Hypertension	**YES**	47	30.1	90	51.1	119	56.9	46	74.2	χ^2=43.27
	NO	109	69.9	86	48.9	90	43.1	16	25.8	P=0.0000
Diabetes	**YES**	5	3.2	16	9.1	42	20.1	23	37.1	χ^2=51.70
	NO	151	96.8	160	90.9	167	79.9	39	62.9	P=0.0000
Hyper-	**YES**	61	39.1	93	52.8	124	59.3	30	48.4	χ^2=15.05
cholesterolemia	**NO**	95	60.9	83	47.2	85	40.7	32	51.6	P=0.0018
BMI≥25kg/m^2	**YES**	59	37.8	99	56.3	130	62.2	44	71.0	χ^2=29.49
	NO	97	62.2	77	43.7	79	37.8	18	29.0	P=0.0000
Smoking	**YES**	52	33.3	54	30.7	63	30.1	12	19.4	χ^2=4.21
	NO	104	66.7	122	69.3	146	69.9	50	80.6	P=0.2396
Alcohol	**YES**	76	48.7	91	51.7	87	41.6	25	40.3	χ^2=5.16
consumption	**NO**	80	51.3	85	48.3	122	58.4	37	59.7	P=0.1603
Physically	**YES**	68	43.6	90	51.1	131	62.7	42	67.7	χ^2=18.30
Active	**NO**	88	56.4	86	48.9	78	37.3	20	32.3	P=0.0004

The prevalence of Hypertension (By history of previously known disease or SBP ≥140 or DBP ≥ 90mmHg) steadily increased with age, in the age group of 20-29 years, it

was 30.1% which increased rapidly and reached a prevalence of 74.2% in the age group of 50-59 years.

The prevalence of Diabetes (By history of previously known disease or fasting plasma glucose of ≥126 mg/dl) steadily increased with age, in the age group of 20-29 years, it was 3.2%. There was sharp increase in prevalence of Diabetes above the age of 39 years and rapidly reached a prevalence of 37.1% in the age group of 50-59 years.

The prevalence of Hypercholesterolemia (Total cholesterol level ≥ 200 mg/dl) steadily increased with age, in the age group of 20-29 years, it was 39.1%. The prevalence of Hypercholesterolemia increased up to 49 years and plateaued thereafter to reach a prevalence of 48.4% the age group of 50-59 years.

The prevalence of Overweight (BMI≥25kg/m^2) individuals steadily increased with age, in the age group of 20-29 years, it was 37.8% which increased rapidly and reached a prevalence of 71.0% in the age group of 50-59 years.

The prevalence of Smoking was roughly similar in all the age groups, with a decline in the prevalence in the age group of 50-59 years.

The prevalence of Alcohol consumption was roughly similar in all the age groups, with a small peak in the prevalence 57.1% in the age group of 30-39 years.

The prevalence of Physical Activity significantly increased with age, in the age group of 20-29 years, it was 43.6% and reached a prevalence of 67.7% in the age group of 50-59 years (probably due to better awareness and advice from company physician).

Prabhakaran et al., observed that diabetes, hypertension, overweight, showed rising trends with age, while the prevalence of smoking, was roughly similar in all the age groups. Even the youngest age group of 20–29 years had a high prevalence of risk

factors such as dyslipidaemia, hypertension and diabetes. The prevalence of hypertension and pre-hypertension increased with age in the multi-centric study.[11] Kaur et al.,[78] in their study found that there was significant increase in prevalence of current smoking, high risk BMI, central obesity, hypertension, diabetes, hypercholesterolemia with increasing age ($p<0.001$).

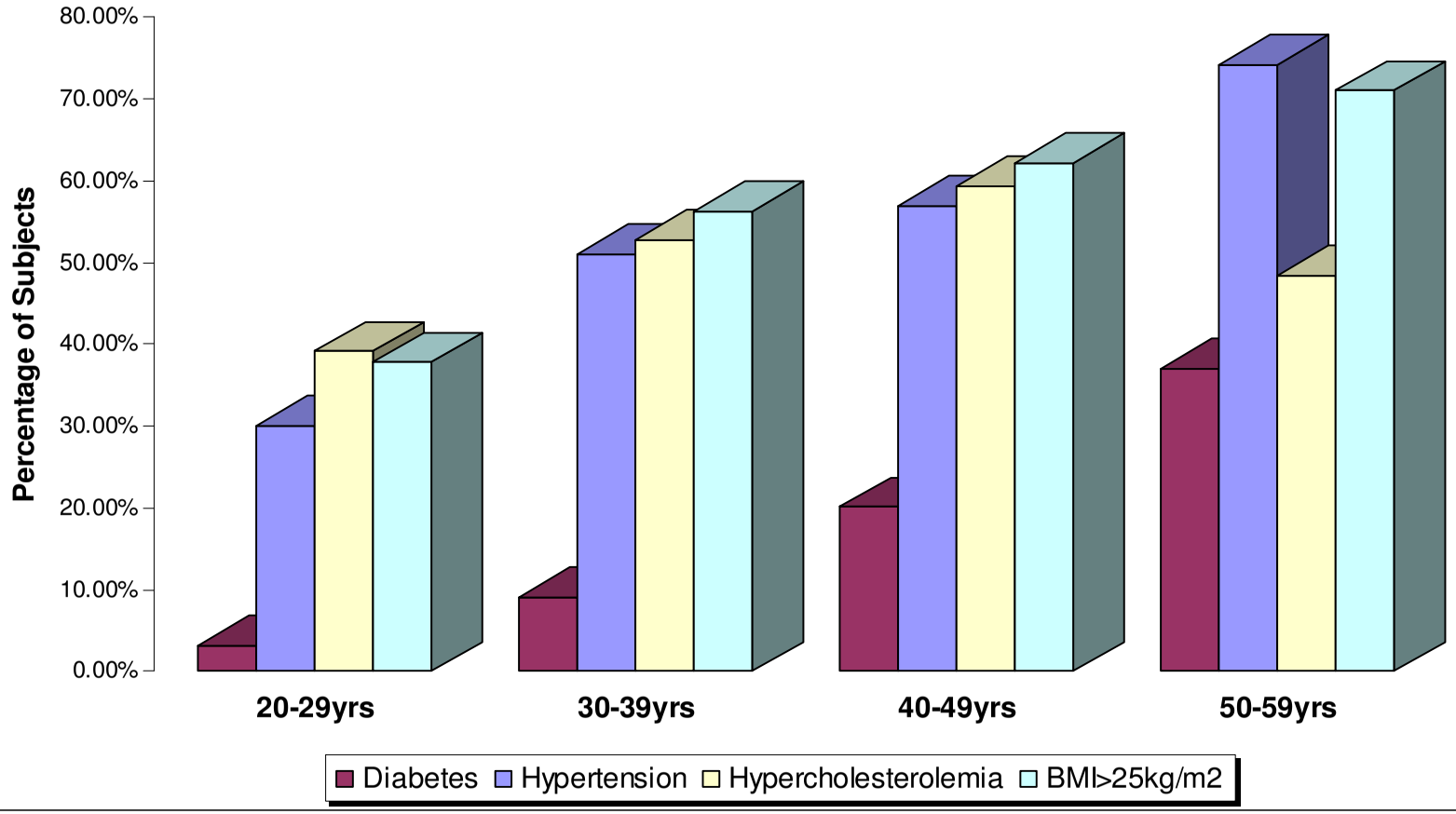
In this study as age increased there was a significant increase in the prevalence of diabetes, hypertension, high risk BMI and hypercholesterolemia as seen in Fig-5.

Table – 18: Mean Values Of CVD Risk Factors By Age n = 603

Cardio-vascular disease risk factor		20-29 years n=156	30-39 years n=176	40-49 years n=209	50-59 years n=62	F – statistic p - value
Systolic Blood Pressure(mmHg)	Mean	124.42	128.07	133.49	140.16	F = 25.21
	SD	12.51	12.36	15.22	15.73	p = 0.0000
Diastolic Blood Pressure(mmHg)	Mean	80.51	84.08	85.90	87.58	F = 14.20
	SD	8.41	8.95	9.31	9.35	p = 0.0000
Fasting Blood Sugar(mg/dl)	Mean	89.88	96.61	109.30	113.10	F = 9.21
	SD	32.68	32.36	50.18	45.84	p = 0.0000
Total Serum Cholesterol(mg/dl)	Mean	194.97	203.84	207.70	204.98	F = 3.45
	SD	34.54	39.99	37.65	42.54	p = 0.0164
Body Mass Index(kg/m^2)	Mean	24.17	25.82	26.08	27.01	F = 15.26
	SD	3.36	3.22	3.37	3.18	p = 0.0000

In this study there was a significant increase in the mean values of CVD risk factors as age increased. Similar finding were reported by Kutty VR et al,[88] in a study conducted in a population in South Kerala.

FIGURE -5: Proportion of study subjects with hypertension, diabetes,hypercholesterolemia and BMI>25kg/m2 across age groups. (n=603)



SECTION – E. EFFECT OF ADIPOSITY ON CVD RISK FACTORS

Table–19: Distribution of CVD Risk Factors According To Body Mass Index(kg/m^2)

n = 603

Cardio-vascular disease risk factor		Under-weight <18.5 n=15		Normal range 18.5–24.9 n=256		Pre-obese 25 – 29.9 n=271		Obese 30 – 40 n=61		Chi-square
		No.	%	No.	%	No.	%	No.	%	
Hypertension	**YES**	1	6.7	107	41.8	150	55.4	44	72.1	χ^2=33.21
	NO	14	93.3	149	58.2	121	44.6	17	27.9	p=0.0000
Diabetes	**YES**	0	0	35	13.7	40	14.8	11	18	χ^2=3.33
	NO	15	100	221	86.3	231	85.2	50	82	p=0.3432
Hyper-	**YES**	5	33.3	124	48.4	144	53.1	35	57.4	χ^2= 4.03
cholesterolemia	**NO**	10	66.7	132	51.6	127	46.9	26	42.6	p=0.2580

There was a continuous graded increase in the prevalence of hypertension with increasing BMI and this was highly significant (Chi-Square for linear trend 30.99 p=0.0000).There was an increase in the prevalence of diabetes and hypercholesterolemia with increasing BMI but this was not statistically significant (see Table-19 & Fig-6).

Prabhakaran et al., reported that there was a continuous graded increase in mean values as well as risk factor prevalence with increasing BMI. Ramachandran et al.,[89] in a study from Chennai observed increasing trend of diabetes at lower BMI. Deshmukh et al.,[90] observed hypertension in rural Indian population at lower BMI.

FIGURE - 6: Proportion of study subjects with hypertension, diabetes and hypercholesterolemia across Body Masss Index. (n=603)

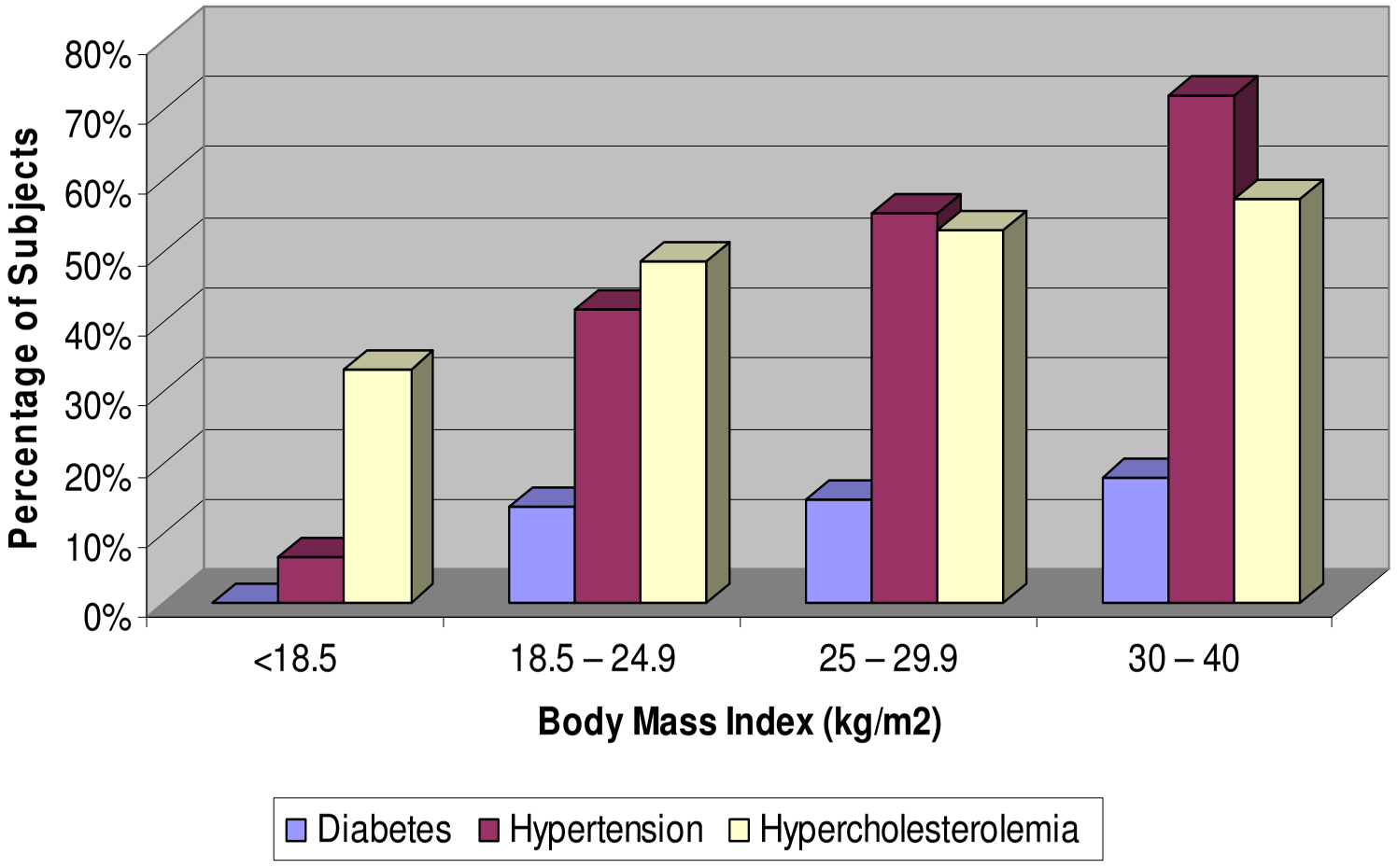


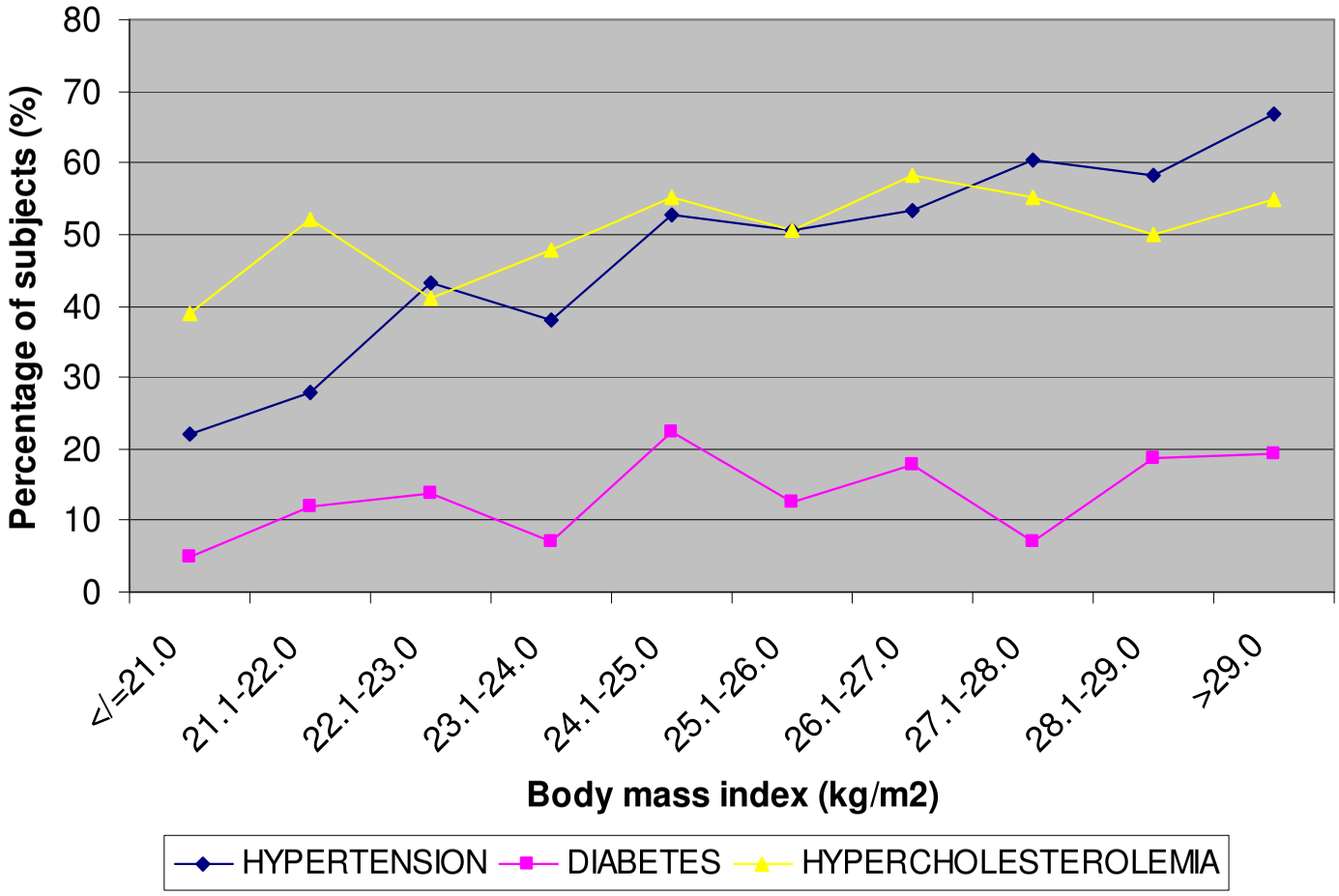
Table – 20: Mean Values Of CVD Risk Factors By BMI (kg/m^2) n =603

Cardio-vascular disease risk factor		Under-weight <18.5 n=15	Normal range 18.5–24.9 n=256	Pre-obese 25 – 29.9 n=271	Obese 30 – 40 n=61	F – statistic p - value
Systolic Blood	Mean	116.67	127.88	131.57	137.71	F = 13.36
Pressure(mmHg)	SD	9.76	14.01	13.62	17.83	p = 0.0000
Diastolic Blood	Mean	76.00	82.34	85.03	89.84	F = 16.77
Pressure(mmHg)	SD	7.37	8.59	8.80	11.03	p = 0.0000
Fasting Blood	Mean	83.80	99.91	104.11	95.56	F = 1.77
Sugar(mg/dl)	SD	10.49	43.26	44.58	19.30	p = 0.1513
Total Serum	Mean	186.67	200.63	204.89	208.59	F = 1.89
Cholesterol(mg/dl)	SD	36.58	36.74	40.33	35.29	p = 0.1285

Among the study subjects, the mean values of SBP and DBP increased significantly as the BMI increased. In the third examination cycle of the Framingham Offspring Study, BMI was significantly and linearly associated with systolic blood pressure, fasting glucose levels, plasma total cholesterol in a study conducted by Lamon-Fava et al.,.[91]

When the BMI is divided into smaller categories with class intervals of 1kilogram/m^2 also the linear trend is obvious, as seen in Fig-7.

FIGURE- 7: Proportion of study subjects with hypertension, diabetes and hypercholesterolemia across body mass index range.(n=603)



Effect Of Smoking And Alcohol Consumption On Selected CVD Risk Factors

There was no significant difference in the prevalence of CVD risk factors among smokers and non-smokers and among alcohol consumers and non-consumers.(Table 21 & 22)

Table – 21: Distribution of CVD Risk Factors According To Smoking Status

Cardio-vascular disease risk factor		Smokers n=181		Non-Smokers n=422		Chi-square
		No.	%	No.	%	
Hypertension	YES	85	47.0	217	51.4	χ^2=1.01
	NO	96	53.0	205	48.6	p=0.3154
Diabetes	YES	28	15.5	58	13.7	χ^2=0.31
	NO	153	84.5	364	86.3	p=0.5786
Hyper-	YES	97	53.6	211	50	χ^2=0.65
cholesterolemia	NO	84	46.4	211	50	p=0.4188

Table – 22: Distribution of CVD Risk Factors According To Alcohol Consumption

Cardio-vascular disease risk factor		Alcohol Consumers n=279		Non-Consumers n=324		Chi-square
		No.	%	No.	%	
Hypertension	YES	144	51.6	158	48.8	χ^2=0.49
	NO	135	48.4	166	51.2	p=0.4856
Diabetes	YES	36	12.9	50	15.4	χ^2=0.78
	NO	243	87.1	274	84.6	p=0.3759
Hyper-	YES	153	54.8	155	47.8	χ^2=2.94
cholesterolemia	NO	126	45.2	169	52.2	p=0.0865

SECTION – F. ASSOCIATION BETWEEN HYPERTENSION AND OTHER RISK FACTORS

Table – 23: Distribution Of CVD Risk Factors According To Hypertension Status

Cardio-vascular disease risk factor		Hypertension		Chi-square
		YES	NO	
BMI≥25kg/m^2	YES	194	138	$\chi^2 = 20.61$
	NO	108	163	p = 0.0000
Diabetes	YES	52	34	$\chi^2 = 4.33$
	NO	250	267	p = 0.0376
Hyper-	YES	156	152	$\chi^2 = 0.08$
cholesterolemia	NO	146	149	p = 0.7812
Smoking	YES	85	96	$\chi^2 = 1.01$
	NO	217	205	p = 0.3154
Alcohol	YES	144	135	$\chi^2 = 0.49$
Consumption	NO	158	166	p = 0.4856
Physical	YES	175	156	$\chi^2 = 2.28$
Activity	NO	127	145	p = 0.1310

Significant relationship of hypertension was observed with high risk BMI (OR: 2.12; 95% CI=1.53 - 2.94; P=0.0000), and Diabetes (OR: 1.63; 95% C.I.=1.03 - 2.60; P=0.0192). No significant relationship could be observed for hypercholesterolemia, smoking, alcohol consumption and physical activity.

SUMMARY

The results of this study indicated a high prevalence of behavioral risk factors (smoking and alcohol consumption), obesity, hypertension, diabetes and hypercholesterolemia among middle and high-income young males in an urban industrial population in Chennai. Prevalence of these risk factors increased during the most productive years putting them at risk of cardiovascular morbidity and mortality at relatively younger age. This population provided an opportunity to study the influence of socioeconomic and lifestyle transition on the prevalence of cardiovascular risk factors. The results were consistent with the multi-centric study done in industrial settings in India.

Study population included 603 males aged 22-59 years. The blood samples were collected for all subjects. Mean age was 37.68 ±9.19years. Nearly one third 209(34.7%) of the participants were in the 40–49 years' age group. All had graduate or post-graduate level education. All were semi-professionals belonging to the upper middle class. Average family income was Rs.11,500 per month (8000-15000).

Tobacco consumption was prevalent in 181(30.0%) subjects and 279(46.3%) of the study subjects were alcohol consumers.

Prevalence of overweight (BMI 25.0 kg/m2-29.9 kg/m2) and obesity (BMI ≥ 30.0 kg/m2) using WHO definition was 44.9%and 10.1% respectively. As per WHO recommendations for defining risk thresholds among Asians, 52.9% were in increased risk (BMI 23.0 kg/m2-27.4 kg/m2) and 26.5% were in high-risk (BMI ≥ 27.5 kg/m2) category.

Pre-hypertension and hypertension were prevalent in 247(41.0%) and 302 (50.1%) subjects respectively. Among the hypertensives, 247 (81.78%) were newly detected. Among known hypertensives, 8(14.54%) had blood pressure under control (SBP <140 mmHg and DBP<90 mmHg).

Prevalence of impaired fasting glucose and diabetes mellitus was 85(14.1%) and 86(14.3%) respectively. Among the diabetics, 28(32.6%) were newly detected. Among known diabetics, 29 (50.0%) had blood glucose under control (<126 mg/dL).

308(51.1%) of the subjects had raised total cholesterol.

There was significant increase in prevalence of high risk BMI, hypertension, diabetes, hypercholesterolemia with increasing age.

Prevalence of hypertension, diabetes and hypercholesterolemia increased across the BMI range. The linear trend was statistically significant for hypertension.

Significant relationship of hypertension was observed with BMI (OR: 2.12; 95% CI=1.53 - 2.94; P=0.0000) and Diabetes (OR: 1.63; 95% CI=1.03 - 2.60; P=0.0192).

No significant relationship could be observed for other risk factors.

RECOMMENDATIONS

Most of us understand that the health or ill-health of workers in an industry is related to the hazards posed by the occupational environment. The effects of Lifestyle diseases are likely to be similar to those outside the working environment. In a way, the industrial worker is 'captive' whose major lifestyle gets adapted to his/her work needs.[92] The rationale of screening these workers for NCD risk factors is not justified unless these activities are strongly linked to continuous and holistic health promotion programs. These issues have been laid down in the technical and ethical guidelines of the International Labour Organization for industrial workers.[93]

Also, in the absence of a population based nationally representative surveillance system for CVD, establishing a multi-centric surveillance system in industrial settings could be a useful initial step.

The high prevalence of risk factors is a cause for concern, organized sector industries provide a unique opportunity for carrying out prevention programmes. It is estimated that there are nearly 6 million people working in such large organized sector industries in India. While most of these have their own primary healthcare facilities, they also provide for the healthcare of employees and their dependents in higher medical institutions when required. With the rising burden of CVD, the expenditure on such healthcare programmes by every industry is likely to increase enormously. Thus, there is a lot of scope for and benefit in initiating comprehensive, low-cost CVD prevention programmes at the workplace for employees and their dependants. Such onsite programmes have been found to be modestly successful in the West through increased

awareness, health education and risk reduction interventions, and modified models could easily be adapted to Indian settings. This is likely to lead to a healthier workforce as well as a decrease in expenditure by the industry on treatment costs and increased absenteeism.

The importance of workplace health promotion was addressed in 1950 and later updated in 1995 in a joint International Labour Organization/World Health Organization session on occupational health.

In response to the global burden imposed by non-communicable diseases, **WHO developed the Global Strategy on Diet, Physical Activity and Health**[94] (DPAS), which was adopted by the 57th World Health Assembly in May 2004. The World Health Organization /World Economic Forum Joint Event was held in Dalian, People's Republic of China, on 5-6 September 2007. The report summarizes the current evidence available in addressing the different dimensions of the workplace as a key setting for interventions designed to prevent NCD through diet and physical activity.

The workplace as a health promotion setting

- this setting can serve to improve the health status of workers and contribute to a positive and caring image of the company.
- improvements in staff morale and productivity, and reductions in staff turnover, absenteeism and sick leave.
- reductions in health plan costs, workers' compensation and disability costs.

- significant proportion of time spent at work by the large majority of the population
- it offers an opportunity to utilize peer pressure to encourage employees to make desirable alterations to their health habits.

Senior managers may have a variety of reasons for wanting healthier employees. It is therefore important to target the reasons and motivations if senior management are to engage successfully in the implementation of Workplace health promotion programmes, and for the programmes to be effective and yield results.

Key elements of successful programmes

The development and implementation of Workplace health promotion programmes should consider the following elements: clear goals and objectives, links with programmes to business objectives, strong management support and effective communication, and supportive environments.

The essential role of a multi-stakeholder approach

Different stakeholders that can play a role in Workplace health promotion include: international organizations; ministries of health, labour and safety; local and municipal governments; nongovernmental organizations; civil society; employers; employees; trades unions; company health insurance funds; the agriculture industry; food producers, catering and food distributors; and the sports industry.

This study reinforces the need for **low-cost workplace intervention** programs. Recommendations were made to the management that included implementation of strict no smoking policy inside the campus, smoking cessation clinic, health education programme to increase awareness about healthy lifestyle, modification of the canteen meal menu and to motivate sedentary employees to participate in sports and other physical fitness programmes.

In addition, **periodic follow-up** of the employees with hypertension, diabetes and hypercholesterolemia in the in-house clinic was recommended. All the employees should undergo an **annual check up**, for early detection of any abnormalities. The management of the factory was also considering a proposal to start a gymnasium and recreational center for the benefit of the employees.

Comprehensive and **integrated** action is the means to prevent and control chronic diseases. There is no single dedicated programme for Non Communicable Diseases. Separate programmes exist such as National Cancer Control Programme, National Diabetes Control Programme, National Programme for Control of Blindness. These can be Integrated and brought together under one programme, modelled like The National Vector Borne Diseases Control Programme (NVBDCP).

At the center, a **Directorate of National Programme for Prevention and Control of Non Communicable Diseases,** the national level Technical Nodal office equipped with Technical Experts in various fields (Public Health, CVDs, Diabetes, Cancer, Respiratory conditions, Blindness, etc.) responsible for framing technical guidelines and policies to guide the states for implementation of Programme strategies, budgeting and planning, monitoring and periodical evaluation.

Regional Offices providing technical advice as well as assistance to the state and responsible for conducting risk factor surveys, cross checking of blood biochemical estimation, capacity building of the states.

State Level is responsible for implementation of Programme with District non-communicable disease units under them.

At the Primary Health Centres early detection and treatment of uncomplicated cases can be done. Complicated cases can be referred to Community Health Centres and other secondary and tertiary level Health institutions.

Strengthen research and Information Education and Communication activities.

LIMITATIONS

1. The results of the study cannot be generalized to the general urban population as the study was done in an industrial population that had higher socio-economic status and unique work profile.
2. In addition, study population essentially consisted of males.
3. A detailed history of diet and stress was not elicited.

REFERENCES

1. Regional Health Forum – Volume 6, Number 1, 2002

2. Eric J Topol, Robert M Califf, Eric N Prystowsky, James D Thomas, Paul D Thompson Textbook of Cardiovascular Medicine, Third Edition

3. Preventing chronic diseases: a vital investment. Geneva, World Health Organization, 2005.

4. http://www.who.int/dietphysicalactivity/publications/facts/cvd/en/

5. Roger Detels, James McEwen, Robert Beaglehole, Heizo Tanaka Oxford Textbook of Public Health, Fourth Edition

6. Bonita R, de Courten M, Dwyer T, Jamrozik K, Winkelmann R. Surveillance of risk factors for noncommunicable diseases: The WHO STEPwise approach. Summary.Geneva, World Health Organization, 2001.

7. World Health Organization. The world health report 2004: Changing history. Geneva: WHO; 2004.

8. World Health Organization. The world health report 1999: Making a difference. Geneva: WHO; 1999.

9. World Health Organization. The world health report 2002: Reducing risks, promoting life. Geneva: WHO; 2002.

10. Gupta R. Burden of coronary heart disease in India. Indian Heart J 2005; 5 : 632-8.

11. Reddy KS, Prabhakaran D, Chaturvedi V, Jeemon P, Thankappan KR, Ramkrishnan L, et al. Methods for establishing a surveillance system for cardiovascular diseases in Indian industrial populations. Bull World Health Organ 2006; 84:460-469.

12. Goyal A, Yusuf S The burden of cardiovascular diseases in the Indian sub continent. Indian J Med Res 2006; 124:235-244.

13. Prabhakaran D, Shah P, Chaturvedi V, Ramakrishnan L, Manhapra A, Reddy KS. Cardiovascular risk factor prevalence among men in a large industry of northern India.The Natl Med J India 2005; 8(2):59-65.

14. Karlehagen S, Ohlson CG.Primary prevention of cardiovascular disease by an occupational health service. Prev Med. 2003 Sep;37(3):219-25.

15. Theorell T, Karasek RA. Current issues relating to psychosocial job strain and cardiovascular disease research. J Occup Health Psychol. 1996 Jan;1(1):9–26.

16. Job characteristics and the incidence of myocardial infarction. N HAMMAR, L ALFREDSSON, T THEORELL International journal of epidemiology 23:22, 277-284

17. Cardiovascular risk factors in American and Japanese executives Telecom Health Research Group (1977) Journal of the Royal Society of Medicine Volume 78 July 1985

18. Effect of Working Hours on Biological Functions related to Cardiovascular System among Salesmen in a Machinery Manufacturing Company Kenji Iwasaki et al Industrial Health 1998, 36, 361-367

19. WHO Technical Series - 1996 – Control of Hypertension

20. R Gupta.Trends in hypertension epidemiology in India. Journal of Human Hypertension (2004) 18, 73–78.

21. Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: analysis of worldwide data. Lancet 2005; 365: 217–23.

22. The ALLHAT Officers and Co-ordinators for the ALLHAT Collaborative Group. Major outcomes in high-risk hypertensive patients randomized to angiotensin converting enzyme inhibitor or calcium channel blocker vs diuretic. The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT). JAMA. 2002;288:1981-1997.

23. Chobanian AV, Bakris GL, Black HR, et al. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003;42:1206-1252.

24. World Health Organization, International Society of Hypertension Writing Group. 2003 World Health Organization (WHO)/International Society of Hypertension (ISH) statement on management of hypertension. J Hypertens. 2003;21:1983-1992.

25. Stamler J. Blood pressure and high blood pressure: Aspects of risk. Hypertension 1991;18 (suppl.):I.95–I.107.

26. Vasan RS, Larson MG, Leip EP, Evans JC, O-Donnell CJ, Kannel WB, ET AL. Impact of high-normal blood pressure on the risk of cardiovascular disease. N Engl J Med 2001, 345:1291–1297

27. Lewington S, Clarke R, Qizilbash N, Peto R, Collins R. Age-specific relevance of usual blood pressure to vascular mortality. Lancet. 2002;360:1903-1913.

28. Whelton PK, He J, Appel LJ, et al. Primary prevention of hypertension: clinical and public health advisory from The National High Blood Pressure Education Program. JAMA. 2002;288:1882-1888.

29. American Diabetes Association Diagnosis and classification of diabetes mellitus. Diabetes Care. 2004 Jan; volume 27 Suppl 1:S5-S10.

30. WHO(1985). Techn. Rep. Ser., No. 727.

31. Jafar TH. The growing burden of chronic kidney disease in Pakistan. N Engl J Med 2006; 354 : 995-7.

32. Reddy KS, Shah B, Varghese C, Ramadoss A. Responding to the threat of chronic diseases in India. Lancet 2005; 366 : 1746-51.

33. ANNIKA M. ADLERBERTH, MD ANNIKA ROSENGREN, MD LARS WILHELMSEN, MD Diabetes and Long-Term Risk of Mortality From Coronary

and Other Causes in Middle-Aged Swedish Men DIABETES CARE, VOLUME 21, NUMBER 4, APRIL 1998

34. Stamler J, Vaccaro O, Neaton JD, Wentworth D: Diabetes, other risk factors, and 12-year cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial. Diabetes Care 16:434-444, 1993

35. Lehto S, Ronnemaa T, Haffner SM, et al (1997) Dyslipidemia and hyperglycemia predict coronary heart disease events in middle-aged patients with NIDDM. Diabetes, 48, 1354 -9.

36. Misra A, Luthra K, Vikram NK. Dyslipidemia in Asian Indians: determinants and significance. J Assoc Physicians India 2004; 52: 137–42.

37. Gupta R, Prakash H, Kaul V. Cholesterol lipoproteins, triglycerides, rural-urban differences and prevalence of dyslipidaemia among males in Rajasthan. J Assoc Physicians India 1997;45:275-9.

38. Goyal A, Yusuf S The burden of cardiovascular diseases in the Indian sub continent. Indian J Med Res 2006; 124:235-244.

39. Roger Detels, James McEwen, Robert Beaglehole, Heizo Tanaka Oxford Textbook of Public Health, Fourth Edition 1135.

40. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 2001; 285:2486-2497.

41. Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attributable to Selected Major Risk Factors By Majid Ezzati, World Health Organization 2004, 391-496.

42. Hager, A. (1981).Br. Med. Bull., 37(3) 287.

43. World Health Organisation, International Association for the Study of Obesity, International Obesity Task Force. The Asia-Pacific Perspective: Redefining obesity and its treatment. Sydney: Health Communications, 2000.

44. World Health Organization. Obesity- Preventing and managing the global epidemic. WHO technical report series 894.Geneva: WHO; 1999.

45. Reddy KS, Prabhakaran D, Shah P, Shah B. Rural-urban differences in distribution of body mass index and waist-hip ratios in north Indian population samples. Obes Rev 2002; 3: 197–202.

46. Gupta R, Gupta VP, Sarna M, Prakash H, Rastogi S, Gupta KD. Serial epidemiological surveys in an urban Indian population demonstrate increasing coronary risk factors among the lower socioeconomic status. J Assoc Physicians India 2003; 51: 470–77.

47. www.who.int/entity/world-health-day/previous/2002/files/whd02_move_for_health_en.pdf

48. Vaz M, Bharathi AV. Practices and perceptions of physical activity in urban, employed, middle-class Indians. Indian Heart J 2000; 52: 301–06.

49. Goyal A, Yusuf S The burden of cardiovascular diseases in the Indian sub continent. Indian J Med Res 2006; 124:235-244.

50. DHHS (Department of Health and Human Service). Physical Activity and Health: A Report of the Surgeon General. Atlanta: National Center for Chronic Disease Prevention and Health Promotion, 1996.

51. Pate RR, Prat M, Blair SN et al. Physical activity and public health:A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. JAMA 1995; 273:402–07.

52. Rastogi T, Vaz M, Spiegelman D, Reddy KS, Bharathi AV, Stampfer MJ, et al. Physical activity and risk of coronary heart disease in India. Int J Epidemiol 2004; 33 : 759-67.

53. Reddy KS, Shah B, Varghese C, Ramadoss A. Responding to the threat of chronic diseases in India. Lancet 2005; 366 : 1746-51.

54. The Health Consequences of Smoking; A Report of the Surgeon General: 1972.

55. Bain C. et al (1978). Lancet 1: 1087

56. The health consequences of smoking: a report of the Surgeon General. (2004)

57. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet 2004; 36 : 937-52.

58. WHO Report on the Global Tobacco Epidemic, 2008 - The MPOWER package

59. WHO(1985) Primary Prevention of CHD EURO Rep and studies 98. Copenhagen.

60. Jürgen Rehm, Ph.D., Gerhard Gmel, Ph.D., Christopher T. Sempos, Ph.D., and Maurizio Trevisan, M.D., M.S. Alcohol-Related Morbidity and Mortality. Alcohol Research & Health. Vol. 27, No. 1, 2002.39-51.

61. Charlotte Turner, Peter Anderson, Alcohol and cardiovascular disease: what is the relationship? Addiction Volume 85 Issue7,851-853

62. www.thefreedictionary.com/employee

63. www.prospect.ac.uk

64. Peter R, Psychosocial work environment and risk of coronary heart disease. Int. Arch. Occupational Health 2000.

65. Robert Karasek Chantal Brisson The Job Content Questionnaire (JCQ): An Instrument for Internationally Comparative Assessments of Psychosocial Job Characteristics Journal of Occupational Health Psychology 1998, Vol. 3, No. 4, 322-355

66. Shri Nilachal Ray, Unorganized vis-à-vis Organized Manufacturing Sector in India.

67. http://mohfw.nic.in/for%20websitediabetes.htm

68. P Kaur, TV Rao, S Sankarasubbaiyan, AM Narayanan, R Ezhil, Sudha Ramachandra Rao, MD Gupte, Prevalence and Distribution of Cardiovascular

Risk Factors in An Urban Industrial Population in South India: A Cross-Sectional Study JAPI • VOL. 55 • NOVEMBER 2007: 771-776.

69. Kaplan's Clinical Hypertension. Measurement of Blood Pressure. 23-34.

70. WHO report on the global tobacco epidemic, 2008

71. Saverio Stranges; Tiejian Wu; Joan M. Relationship of Alcohol Drinking Pattern to Risk of Hypertension A Population-Based Study Hypertension. 2004;44:813-819

72. Chadha SL, Gopinath N, Shekhawat S. Urban–rural differences in the prevalence of coronary heart disease and its risk factors in Delhi. Bull World Health Organ 1997;75:31–8.

73. Ramachandran A, Snehalatha C, Latha E, Satyavani K, Vijay V. Clustering of cardiovascular risk factors in urban Asian Indians. Diabetes Care 1998;21:967–71. Joseph A, Kutty VR, Soman CR. High risk for coronary heart disease in

74. Thiruvananthapuram city: A study of serum lipids and other risk factors. Indian Heart J 2000;52:29–35.

75. Misra A, Pandey RM, Devi JR, Sharma R, Vikram NK, Khanna N. High prevalence of diabetes, obesity and dyslipidaemia in urban slum population in northern India. Int J Obes Relat Metab Disord 2001;25:1722–9.

76. Enas EA, Garg A, Davidson MA, Nair VM, Huet BA, Yusuf S. Coronary heart disease and its risk factors in first-generation immigrant Asian Indians to the United States of America. Indian Heart J 1996;48:343–53.

77. Bhatnagar D, Anand IS, Durrington PN, Patel DJ, Wander GS, Mackness MI, et al. Coronary risk factors in people from the Indian subcontinent living in west London and their siblings in India. Lancet 1995;345:405–9.

78. P Kaur, TV Rao, S Sankarasubbaiyan, AM Narayanan, R Ezhil, Sudha Ramachandra Rao, MD Gupte, Prevalence and Distribution of Cardiovascular Risk Factors in An Urban Industrial Population in South India: A Cross-Sectional Study JAPI • VOL. 55 • NOVEMBER 2007: 771-776.

79. Mehan MB, Srivastava N, Pandya H Profile of non communicable disease risk factors in an industrial setting J Postgrad Med September 2006 Vol 52 Issue 3.

80. Gupta R, Gupta VP, Sarna M, Bhatnagar S, Thanvi J, Sharma V, et al. Prevalence of coronary heart disease and risk factors in an urban Indian population: Jaipur Heart Watch-2. Indian Heart J 2002;54:59–66.

81. Mohan, D., Sharma, H. K., Darshan, S., Sundaram, K. R. and Neki, J. S. (1978) Prevalence of drug abuse in young in rural Punjab. Indian Journal of Medical Research 68, 689–694.

82. Chakravarthy, C. (1990) Community workers estimate of drinking and alcohol-related problems in rural areas. Indian Journal of Psychological Medicine 13, 49–56.

83. Mohan, D., Chopra, A., Ray, R. and Sethi, H. (2001) Alcohol consumption in India: a cross sectional study. In Surveys of Drinking Patterns and Problems in Seven Developing Countries, Room, R., Demers, A., Bourgault, C. eds, pp. 103–114. World Health Organization, Geneva.

84. Gupta R, Gupta VP, Ahluwalia NS. Educational status, coronary heart disease, and coronary risk factor prevalence in a rural population of India *British Medical Journal*, 1994, 309:1332-1336

85. Patterns and predictors of prehypertension among "healthy" urban adults in India. Chokalingam A. Ganesan N., Venkatesan S. et al., Angiology 2005 Sep-Oct;56(5):557-63.

86. Prabhakaran D, Shah P, Chaturvedi V, Ramakrishnan L, Manhapra A, Reddy KS. Cardiovascular risk factor prevalence among men in a large industry of northern India.The Natl Med J India 2005; 8(2):59-65.

87. Hazarika NC, Biswas D, Narain K, Kalita HC, Mahanta J. Hypertension and its risk factors in tea garden workers of Assam. Natl Med J India. 2002 Mar-Apr;15(2):63-8.

88. Kutty VR, Soman CR, Joseph A, Kumar KV, Pisharody R. Random capillary blood sugar and coronary risk factors in a south Kerala population. J Cardiovasc Risk. 2002 Dec;9(6):361-7.

89. Snehalatha C, Viswanathan V, Ramachandran A. Cutoff values for normal anthropometric variables in Asian Indian adults. Diabetes Care 2003;26:1380–84.

90. Deshmukh PR, Gupta SS, Dongre AR, Bharambe MS, Maliye C, Kaur S, et al. Relationship of anthropometric indicators with blood pressure levels in rural Wardha. Indian J Med Res 2006;123:657-64.

91. Stefania Lamon-Fava; Peter W.F. Wilson; Ernst J. Schaefer Impact of Body Mass Index on Coronary Heart Disease Risk Factors in Men and Women The Framingham Offspring Study **Arteriosclerosis, Thrombosis, and Vascular Biology.**1996;16:1509-1515.

92. Mathur P Risk factors for non-communicable diseases: Getting beyond data J Postgrad Med September 2006 Vol 52 Issue 3: 171-172.

93. Technical and Ethical guidelines for workers health surveillance. Occupational and Health Series No. 72. International Labour Organization: 1998.

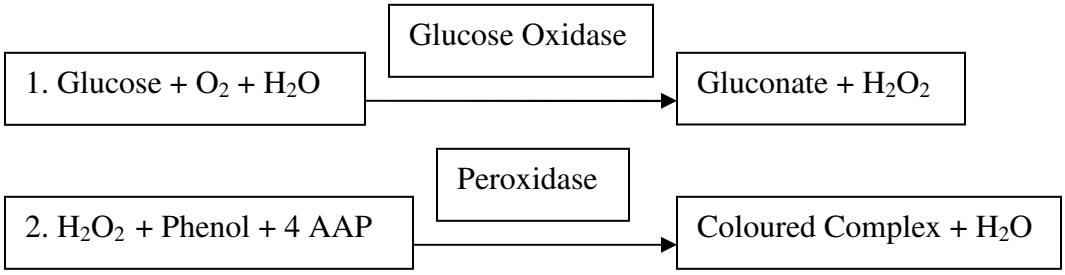
94. Preventing noncommunicable diseases in the workplace through diet and physical activity : WHO/World Economic Forum report of a joint event. World Health Organization / World Economic Forum 2008.

ANNEXURE -2

GLUCOSE ESTIMATION BY GLUCOSE OXIDASE-PEROXIDASE METHOD

Principle

Glucose is oxidized by Glucose Oxidase (GOD) into Gluconic Acid and Hydrogen Peroxide. Hydrogen Peroxide in presence of Peroxidase (POD) oxidizes the chromogen 4-Aminoantipyrine (4 AAP) / phenolic compound to a red coloured compound. The intensity of the red coloured compound is proportional to the glucose concentration and is measured at 505 nm (490-530 nm). The final compound is stable for 2 hours.



Specimen collection

Plasma is preferred for glucose determination, but serum separated immediately after clotting can also be used. The addition of Sodium Fluoride is recommended to inhibit glycolysis. If however this anticoagulant is not used, plasma separation should be carried out within 30 minutes of blood sample collection. As far as possible, samples should be used on the same day. However, if fluoride with any other anticoagulant is used, then the samples are stable for 24hours at room temperature and for 1 week at 2- 8 degree Celsius.

Reagent (Buffer/Enzymes/Chromogen)

Phosphate buffer
4-Aminoantipyrine (4AAP)
p-Hydroxy Benzoic acid
Glucose Oxidase
Peroxidase

Standard

Glucose

Reaction Parameters

Type of Reaction	:	End Point
Wavelength	:	505 nm (490-530 nm)
Incubation	:	15 min, at 37° C
Standard Concentration	:	100 mg/dl
Sample Volume	:	10 microlitres(0.01ml)
Reagent Volume	:	1.0 ml
Zero Setting with	:	Reagent Blank
Light Path	:	1.0 cm

Procedure

Pipette into test tubes	Blank	Standard	Test
Reagent (ml)	1.0	1.0	1.0
Standard (ml)	-	0.01	-
Sample (ml)	-	-	0.01

Mix well and incubate for 15 minutes, 37° C. Read absorbance of standard and test at 505 nm (490-530 nm or with green filter) against reagent blank.

Test Results

$$\text{Glucose Concentration (mg/dl)} = \frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \times 100$$

Linearity

This method is linear up to 500mg/dl.

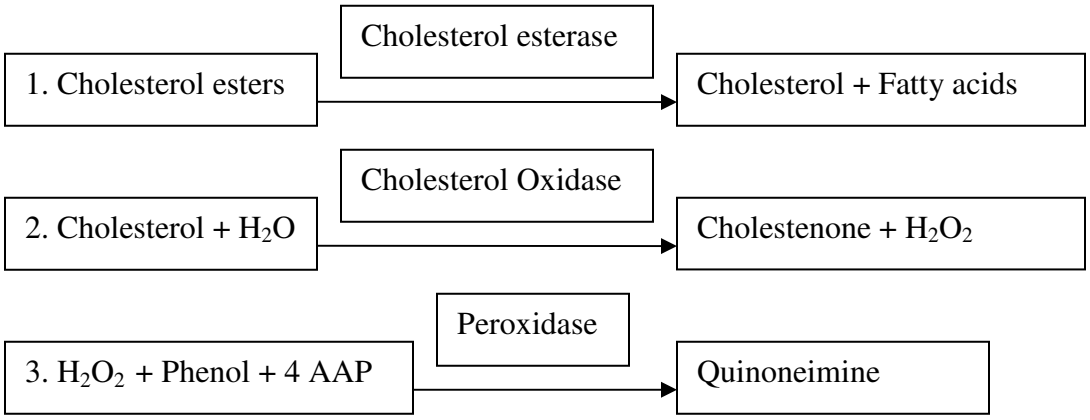
References

- TINDER P, Annual Clinical Biochemistry 6, 24-25 (1969)
- TIETZ N.W. Fundamentals of Clinical Chemistry, 2nd edition W.B. Saunders Co. (1982)

CHOLESTEROL ESTIMATION BY CHOD-PAP METHOD

Principle

The estimation of cholesterol involves the following enzymatic reactions



The intensity of the coloured complex produced is directly proportional to the concentration of Cholesterol content which is measured at 505 nm or with green filter.

Presentation

1. Cholesterol (Enzymes Chromogen Buffer)
2. Cholesterol (Buffer Solution)
3. Cholesterol Standard 200mg/dl

Specimen Collection

Fasting fresh, clear unhemolysed serum is the specimen of choice. Plasma prepared with anticoagulants such as heparin may be used.

Reaction Parameters

Type of Reaction	:	End Point
Sample Volume	:	10 microlitres(0.01ml)
Reagent Volume	:	1.0 ml
Wavelength	:	505 nm (490-530 nm)
Incubation	:	10 min, at 37° C
Light Path	:	1.0 cm
Zero Setting with	:	Reagent Blank

Procedure for colorimeter

Pipette into test tubes	Blank	Standard	Test
Reagent (ml)	2.5	2.5	2.5
Standard (ml)	-	0.02	-
Sample (ml)	-	-	0.02

Mix well and incubate for 10 minutes, 37° C. Read absorbance of standard and test at 505 nm (490-530 nm or with green filter) against reagent blank within 30 minutes.

Test Results

$$\text{Cholesterol Concentration (mg/dl)} = \frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \times 200$$

Linearity

This method is linear up to 700mg/dl.

References

- ROESCHCLAUP et al methods of enzymatic Analysis 2nd Edition, H.O. Bergmeyer, Academic press New York (1074) 1890
- FLEGG H.M. Annals of Clinical Biochemistry. 10-79 (1973)

ANNEXURE-1

QUESTIONNAIRE

A CROSS-SECTIONAL STUDY OF SELECTED MODIFIABLE CARDIOVASCULAR RISK FACTORS AMONG SALES EXECUTIVES IN AN URBAN ORGANIZED SECTOR INDUSTRIAL POPULATION IN CHENNAI

PART 1-DEMOGRAPHIC CHARACTERISTICS

1 NAME

2 AGE

3 SEX

4 EDUCATION

- ☐ Professional, P.G
- ☐ B.A.,B.Sc., Degree
- ☐ Intermediate, High School Diploma
- ☐ High School
- ☐ Middle school
- ☐ Primary school
- ☐ Illiterate

5 OCCUPATION

- ☐ Professional
- ☐ Semi-Professional
- ☐ Clerk/Shopkeeper
- ☐ Skilled Worker
- ☐ Semi-Skilled Worker
- ☐ Unskilled
- ☐ Unemployed

6 FAMILY INCOME PER MONTH

7 SOCIOECONOMIC STATUS

8 MARITAL STATUS

- ☐ Married
- ☐ Unmarried
- ☐ Single

PART 2 - BEHAVIOURAL CHARACTERISTICS

1 **H/O SMOKING**

a. Do you currently smoke any cigarettes?^ ☐ YES ☐ NO ☐ Ex-Smoker

b. If Smoker or Ex-Smoker,On an average how many cigarettes do/did you smoke per day? ☐ < 10 ☐ >10 cigarettes

c. If Smoker or Ex-Smoker, how long have you or had you been smoking? _____ years

d. If Ex-Smoker when did you stop smoking? _____ years

e. Do you consume any other tobacco products? ☐ YES ☐ NO

f. If yes, what do you consume? _____

2 **H/O ALCOHOL CONSUMPTION**

a. Do you currently consume alcohol?* ☐ YES ☐ NO ☐ Past Consumer

b. If Consumer or Ex-Consumer, how frequently do/did you have at least one alcoholic drink? ☐ Regularly ** ☐ Occassionally***

c. If Consumer or Ex-Consumer, how long have you or had you been drinking? _____ years

d. What type of drink do/did you consume? ☐ Beer ☐ Wine ☐ Whiskey/Rum/Brandy

e. How many drinks do/did you consume per day? ☐ upto 2 ☐ 3 to 4 ☐ 5 or more drinks

f. If Ex-Consumer when did you stop drinking? _____ years

5 **H/O EXERCISE**

a. Do you exercise apart from your daily activities? ☐ YES ☐ NO

b. If yes, what type of exercise do you do?
☐ walking
☐ jogging
☐ swimming
☐ gymnasium
☐ others _____

c. How many days in a week do you exercise? ☐ 1to2 ☐ 3to4 ☐ 5to7 days

d. How long do you exercise per day? ☐ < 30 min ☐ 30min or more

6 **DIET** ☐ Vegetarian ☐ Non-Vegetarian

7 **H/O HYPERTENSION**

a. Have you been previously diagnosed to have high Blood Pressure? ☐ YES ☐ NO

b. If yes, are you on medication? ☐ YES ☐ NO

c. Do you take your medication regularly? ☐ YES ☐ NO

8 **H/O DIABETES**

a. Have you been previously diagnosed to have Diabetes? ☐ YES ☐ NO

b. If yes, are you on medication? ☐ YES ☐ NO

c. Do you take your medication regularly? ☐ YES ☐ NO

PART 3 - CLINICAL AND BIOCHEMICAL MEASUREMENTS

1 **ANTHROPOMETRIC MEASUREMENTS**

a. HEIGHT ☐ cms

b. WEIGHT ☐ kgs

c. BMI ☐ kg/m2

2 **BLOOD PRESSURE** ☐ mmHg

3 **BIOCHEMICAL MEASUREMENTS**

a. FASTING BLOOD SUGAR ☐ mg/dl

b. SERUM CHOLESTEROL ☐ mg/dl

THANK YOU

^Smoked at least one cigarette during the last 30 days
*Consumed at least one alcoholic beverage in the past 30 days
**Alcohol intake for more than three times (average) a week.
***Alcohol intake for more than three times a month, but less than three times a week.